THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station — Beltsville, Maryland
Volume 37 — September 15, 1953 — Number 9

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NOTE TO READERS: Please be sure to notify the Survey to take your name off the mailing list if you no longer have need for the Reporter. The mailing list is becoming so large that we must be sure that each copy goes where it will receive maximum use.

PLEASE NOTE: Copy for illustrations for the Reporter should be sent unmounted (See PDR 36(5): 214. May 15, 1952).

POTATO WART ERADICATION PROJECT IN PENNSYLVANIA

R. A. Hyre

The writer visited Dr. R. E. Hartman at Hazelton, Pennsylvania, on July 14-16 in furtherance of the policy of having a representative of the U. S. Department of Agriculture, Bureau of Plant Industry, Soils, and Agricultural Engineering observe different phases of the Pennsylvania Department of Agriculture potato wart (Synchytrium endobioticum) eradication project.

The eradiction program is proceeding according to schedule. The care given to detail inspires confidence that the program will be successfully completed on schedule. Any new infestations discovered from now on will be given total destruction. The plan is for copper sulfate to be applied the first year and the land kept clean and cultivated frequently; an equal amount of lime applied the second year and the land again cultivated frequently; the third year several varieties of vegetables will be grown; and the fourth year potatoes will be grown to demonstrate that the land is fully restored to agricultural use. This procedure has proven successful in limited tests.

The accompanying tabulation is a revised wart eradication summary kindly supplied by Dr. Hartman.

PENNSYLVANIA DEPARTMENT OF AGRICULTURE

POTATO WART ERADICATION SUMMARY

(Compiled by R. E. Hartman)

I WART INFESTED AREAS - 1918 to 1952

Infested Gardens	1,121		
Towns & Villages with Infestations	108		
Counties with Infestations	15		

II WART ERADICATION - 1952 to 1953

Gardens Eradicated:			
Mining Operations	165		
Total Destruction	217		
Standard Eradication	188		
Spot Treatment Eradication	63	633	
Gardens Under Eradication:			
Check Planting:			
Standard Eradication	137		
Spot Treatment Eradication	275		
Under Treatment (Spot Treatment)	76	488	1,121

III CHECK PLANTINGS - 1953 to 1957

Year	Gardens Under Check
1953	488
1954	457
1955	406
1956	319
1957	204

WART ERADICATION SUMMARY 1932 - 1958		
Total Number of Gardens		1,121
Mine Operations	165	
Total Destruction	217	
Standard Eradication	325	
Spot Treatment	414	1,121

WART ERADICANTS 1932 - 1953

Ammonium Thio-cyanate	50,000 Lbs
Copper Sulphate	665,000 Lbs
40% Formaldehyde	174 Bbls

ESTIMATE 1954 - 1957

Copper Sulphate	85,000 Lbs
40% Formaldehyde	100 Bbls

U. S. DEPARTMENT OF AGRICULTURE, DIVISION OF MYCOLOGY AND DISEASE SURVEY, NEWARK, DELAWARE

POTATO TUBER GLASSY-END AND JELLY-END ROT IN THE NORTHEAST IN 1949 AND 1952

B. A. Friedman and Donald Folsom[1]

History

Pethybridge (7) studied "watery or glassy rot", which was prevalent in 1916 in Ireland. Affected tissues were ordinarily sterile, but in some cases Pethybridge isolated Rhizoctonia solani, Botrytis cinerea, and Rhizopus nigricans. He was unable to reproduce the disease by inoculation. Pethybridge reported that glassy rot bore some resemblance to the wet rot, described earlier by Frank (3), in which diseased tissues in the basal end of long and proliferating tubers were soft, watery, and translucent and resembled a cooked turnip. The cells in affected tissue were reported by Frank to have a clear sap and little protoplasm or starch and to die finally with a loss of turgor. Affected tissue was found to contain mycelium of Rhizoctonia solani.

In 1922, Shapovalov (9) stated that the flesh of tapering stem ends of elongated Netted Gem and Burbank potato tubers sometimes differed from the flesh in other parts of the tubers, appearing watery and somewhat translucent and being very deficient in starch. The starch-deficient tissue might remain free of any infection, and then shrivel up with no decomposition, or it might become permeated by Rhizoctonia solani, which did not extend into normal tissue. If Fusarium penetrated the starch-deficient tissue, it sometimes advanced into normal tissue and caused storage rot.

In 1929, Penman (6) described "glassy end" of potatoes in Australia. He reported that the affected flesh was more or less translucent, somewhat darker than normal and characterized by very low starch content. It usually occurred at the stem end but sometimes extended nearly through the tuber. Large tubers and tubers with second growth were affected more than small round ones. No increase in affected tissue or reversion to normal condition occurred in storage but badly affected tubers tended to rot. The trouble was attributed to the translocation of starch into new tuber growth such as develops when growth is resumed after it has been checked by very dry weather.

Fischer (2) in 1930 reported glassy, starch-deficient flesh in the distal or eye-end part of tubers in Austria, especially in elongated new-growth protuberances, and attributed the trouble to unfavorable environmental conditions.

Störmer (10) in Germany described a glassiness in some parts of the tuber, especially the stem end, attributed to starch removal due to renewed growth -- new stolons, tubers, and outgrowths -- in dry seasons.

Schlumberger (8) in Germany in 1934 described "glassy potatoes" as those having the older end poor in starch and looking glassy to the naked eye, caused by second growth after a drought, and drying in storage if they were not invaded by soft rot bacteria in the field.

Greisenegger and Neudecker (4) in Austria in 1935 stated that when drought was followed by rain, the starch in the first generation of tubers was used by new tubers, being reduced to 4.2 percent, and that the depleted tubers were left glassy and worthless.

Murphy (5) in Ireland in 1936 described some effects of drought on potato tubers. Glassy-stem-end occurred in certain long-tubered varieties, following drought, and was associated with second growth and premature sprouting. The glassy flesh was devoid of starch, sloughed off as though cut off by a knife, and contained no parasite capable of rotting normal tubers.

In 1949, Blodgett and Rich (1) classified "jelly end" as a non-parasitic disease which is general over the Northwest, is much more common on long, narrow, or pointed tubers, and is characterized by a jelly-like mass of tissue at the stem end. It extends an inch or more down the tuber and either shrivels and dries, leaving a clear-cut division, or is followed by a Fusarium rot.

Other references might be cited, but they do not distinguish between an initial non-parasitic phase (jelly-end, glassy-end) and the jelly-end rots caused by fungi and bacteria invading the stem end more or less in association with the non-parasitic phase.

[1]Plant pathologists, Bureau Plant Industry, Soils, and Agricultural Engineering, U. S. Department of Agriculture and Maine Agricultural Experiment Station, respectively.

Observations in Maine

In a sample of tubers from the 1949 crop in southwestern Maine the first instance of glassy-stem-end on record in the State was found. In most of the potato-producing area of Aroostook County there was no trouble in the 1952 crop from glassy-end, but from the western and southern parts of this area to the southwestern corner of the State the growing season of 1952 was characterized by rainfall light in July and heavy in August[2] and glassy-end tubers were observed. On Highmoor Farm in southwestern Maine, Green Mountain tubers had eye-end sprouts, nipples, and stolons, the last-named producing a second setting of tubers, and Kennebec tubers grew slender with tapering stem end, but no glassy-end was observed in that variety. In January 1953 a brief survey of stored potatoes in central Maine disclosed glassy-stem-end in slender tapering-end tubers of several Kennebec stocks (Fig. 1). A sample of round Ontario tubers with glassy-stem-end in most of them (Fig. 2, A) and a similar breakdown on other parts of some tubers was received from southern Aroostook County. A sample of round Katahdin tubers with glassy breakdown in various parts of the tuber (Fig. 2, B) was received from the western part of the Aroostook County potato area.

FIGURE 1

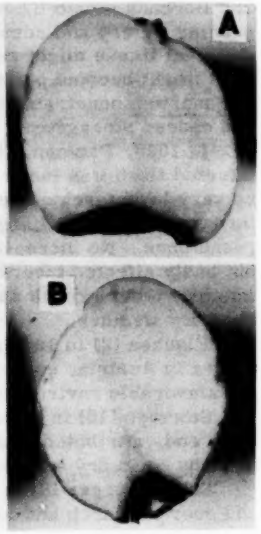

FIGURE 2

FIGURE 1. Kennebec tubers with glassy stem-end characterized by starch-free tissue.
FIGURE 2. A, Ontario tuber, and B, Katahdin tuber, with most of starch-free "glassy" tissue broken down leaving very little debris.

Isolations from the glassy, starch-free tissue of tubers representing all samples were attempted on potato-dextrose agar plates. Seventeen plates were sterile and 25 yielded 9 cultures of fungi among which were Colletotrichum atramentarium, Nigrospora, Penicillium, Trichoderma, and Verticillium albo-atrum and 23 cultures of bacteria. The bacteria were not identified but all produced a shallow wet rot on potato slices, resembling that often caused by Pseudomonas fluorescens. Isolations were attempted also from the dark broken-down tissue that had replaced the glassy tissue. Such tissue was small in volume because there was no starch to give body to it. Four of the agar plates were sterile and 7 yielded 5 cultures of fungi among which were Alternaria tenuis, Phoma tuberosa, and Verticillium albo-atrum and 4 cultures of bacteria which produced a shallow wet rot on potato slices like that produced by

[2]See precipitation maps in Plant Dis. Reptr. 36: 373 and 392.

Pseudomonas fluorescens.

Observations in New York (Long Island Area), New Jersey, and Pennsylvania

The 1949 and 1952 growing seasons in the potato-growing districts of Long Island, New York, New Jersey, and Pennsylvania were characterized by hot, dry weather followed by ample rainfall.

In 1949, samples of tubers affected with jelly- or glassy-end and jelly-end rot were received for identification from inspectors stationed in Long Island and the Hightstown area of New Jersey. Glassy-end tissue was found usually to be sterile, whereas Rhizoctonia, Fusarium and several types of bacteria were isolated from jelly-end rots. By inoculation these organisms were found incapable of reproducing the disease symptoms.

During the 1952 season, several samples of affected lots of tubers from Long Island and New Jersey as well as one lot from Pennsylvania were examined. Tissues at the stem-end of glassy-end tubers were colorless, translucent, and jelly-like in consistency. Affected tissue was usually 1/4 to 1/2 inch deep, but in some cases it was about 1 1/2 inches. At digging time many of the affected stem ends sloughed off, leaving a clear-cut separation. At the same time other tubers in which the jelly-like tissue was either partially shriveled (Fig. 3), or completely shriveled with a dried brown covering of skin, were found. Often at harvest, but more frequently in storage, glassy-end tissue (Fig. 4) was found to be softer than usual, to be discolored light to deep brown, and to be infected with bacteria, Fusarium, or Rhizoctonia. These organisms failed to reproduce the disease in normal tubers upon inoculation. The jelly-end rot seen in eastern-grown tubers resembles the condition which the senior author has observed almost every year in occasional samples of Russett Burbank potatoes from Idaho and in a few cases in White Rose tubers grown in California. The failure to see samples of tubers with physiological jelly-end as described by Blodgett and Rich (1) may be attributed to the breakdown of jelly-end tissue of western-grown potatoes into jelly-end rot during the handling and transit period.

FIGURE 3. Long Island-grown potatoes, variety unknown, showing varying degrees of glassy or jelly end found during harvest.

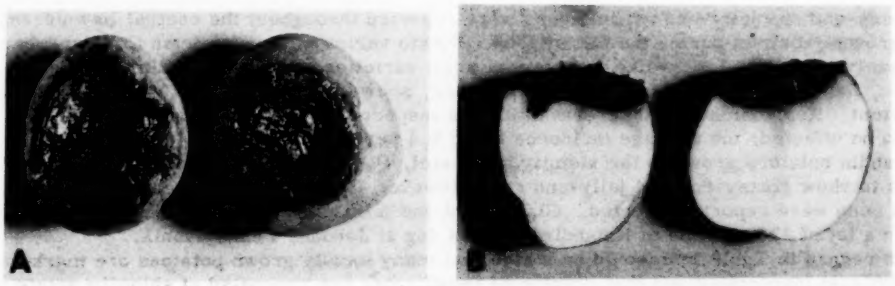

FIGURE 4. Jelly-end rot found at harvest in Long Island-grown tubers, variety unknown; uncut A, cut B.

In addition to containing tubers with definite symptoms of glassy-end and jelly-end rot, some samples of Long Island tubers that appeared normal externally were found to be very watery throughout the flesh when cut and mushy when cooked. R. C. Wright reported that some samples showed very little starch and others no starch when tested with iodine; they were low in total solids and when processed into chips the color was poor and the yield low. In a field near Hicksville, New York, where a sample of watery potatoes was found there were also considerable glassy-end and jelly-end rot, knobbiness, pointed ends, secondary sprouting, and tuber formation. These conditions are often associated with dry weather followed by a rainy spell.

To obtain further information on the prevalence of glassy-end and jelly-end rot during the latter part of 1952, some key inspectors were questioned and inspection certificates issued at some representative cities (Atlanta, Baltimore, Philadelphia, Pittsburgh, and Boston) by the the Fresh Fruit and Vegetable Branch of the Production and Marketing Administration from July 1 to December 31, 1952, were examined.

Regarding this condition in Long Island-grown potatoes, it was stated that "on some days during digging time one-half of the lots grown at the west end of the Island failed to grade because of jelly-end and jelly-end rot," or when such affected tubers were stored in bins "they melted away and standing water accumulated on the floor." It may be seen in Table 1 that glassy-end and jelly-end rot were reported in 23.3 percent of the 90 lots of tubers for which certificates were requested at the five terminal markets, with an average of 2.5 percent of the tubers in the affected lots having the jelly-like condition.

Table 1. Glassy-end and jelly-end rot in eastern-grown potato tubers reported in inspection at some terminal markets, 1952 crop.

Origin	Inspection certificates	Potatoes inspected	Glassy-end and jelly-end rot reported		
			Inspection certificates		Affected tubers
	Number	Tons	Number	Percent	Percent by weight
Long Island, N. Y.	90	1310	21	23.3	2.5
New Jersey	82	1192	33	40.2	5.4
Pennsylvania	26	346	0	0	0
Maine	208	3821	1	0.5	2.0

Glassy-end and jelly-end rot in tubers were reported throughout the central New Jersey potato-growing district during the fall of 1952. Potato varieties such as Irish Cobbler, harvested early, showed no jelly-like symptoms, while varieties such as Katahdin, Chippewa, and Kennebec, harvested during September and October, showed glassy-end and jelly-end rot to some extent. Approximately 40 percent of 82 lots inspected at terminal markets were reported to be affected, the average incidence being 5.4 percent.

Katahdin potatoes grown in the vicinity of Bristol, Pennsylvania, were reported by inspectors to show glassy-end and jelly-end rot. However, in 26 lots inspected at terminal markets none were reported affected. Glassy-end and jelly-end rot were seen by the senior author in a lot of 1200 pounds of Katahdin potatoes dug at Jordan, Pennsylvania.

With regard to Table 1 it should be stated that many locally grown potatoes are marketed without official inspections and that usually no certificates are requested at shipping points where potatoes fail to grade U. S. No. 1 because of glassy-end and jelly-end rot. In addition, there is reason to believe that glassy-end and jelly-end rot were present in some lots in which slimy soft rot (bacterial soft rot) and Fusarium rot were reported. On the other hand, it should not be inferred that about 40 and 23 percent, respectively, of New Jersey and Long Island, New York, lots of potatoes arriving at the five terminal markets would be affected with

glassy-end and jelly-end rot inasmuch as Federal inspections are usually requested for potato shipments in poor condition when there is a question of acceptance or rejection of the shipment or an adjustment of price is sought.

Literature Cited

1. Blodgett, E. C., and A. E. Rich. 1949. Potato tuber diseases, defects, and insect injuries in the Pacific Northwest. Wash. Agr. Expt. Sta. Pop. Bul. 195.
2. Fischer, R. 1930. Glasige Kartoffeln. Oesterr. Zeitschr. f. Kartoffelbau 1930: 81-82. (Abst. in Rev. Appl. Myc. 10: 335).
3. Frank, A. B. 1897. Ueber die Ursachen der Kartoffelfäule. Centralbl. Bakt., Abt. 2., 3: 13-17.
4. Greisenegger, I. K., and __ Neudecker. 1935. Wie können ungewöhnlich stärkearme (glasige) Kartoffeln verwertet werden? Aussergewöhnliche Beschaffenheit von Kartoffelknollen -- eine Folgeerscheinung der abnormen Sommerwitterung. Die Landeskultur. Wien. 2: 10-14. (Abst. in Neuh. Geb. Pflanzensch. 28: 153).
5. Murphy, P. A. 1936. Some effects of drought on potato tubers. Empire Jour. Exp. Agric. 4: 230-246.
6. Penman, F. 1929. "Glassy end" of potatoes. Jour. Dept. Agric. Victoria 27: 450-459. (Abst. in Amer. Pot. Jour. 6: 363 and in Rev. Appl. Myc. 9: 126).
7. Pethybridge, G. H. 1916. Investigations on potato diseases. (Seventh report). Jour. Dept. Agric. and Tech. Instruct. Ireland 16: 564-596.
8. Schlumberger, __. 1934. Glasige Kartoffeln. Die Kartoffel 14: 167-168. (Abst. in Neuh. Geb. Pflanzensch. 28: 84).
9. Shapovalov, M. 1922. Rhizoctonia solani as a potato-tuber rot fungus. Phytopathology 12: 334-336.
10. Störmer, Stettin. 1934. Die Glasigkeit der Kartoffel. Der Kartoffelbau 18: 109-110.

U. S. DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, NEW YORK, NEW YORK AND MAINE AGRICULTURAL EXPERIMENT STATION, ORONO, MAINE

OCCURRENCE OF POTATO Y VIRUS ON TOMATOES IN FLORIDA[1]

Robert A. Conover and Robert W. Fulton

Since 1947 two virus diseases have been observed in tomato fields in Dade County, Florida, that differ in symptomatology from other common viroses of the crop. Specimens of diseased plants were sent to the junior author for identification of the viruses present. Potato Y virus (veinbanding) was found in both diseases. In one, it was the only virus recovered; in the other, tobacco mosaic virus was also present. This paper presents descriptions of the diseases with evidence identifying the viruses concerned.

Potato Y virus has not received much attention as the incitant of a field disease of tomato. Johnson[2] in 1930 recorded natural infection of tomato in Kentucky. Smith[3] reported that the virus was occasionally found affecting tomatoes in England. Sakimura[4] found Y virus associated with spotted wilt virus in Hawaiian tomatoes. There are many references to the susceptibility of the tomato following artificial inoculation. These records of potato Y virus in tomato have indicated a mild disease -- a faint mottle, slight distortion, and veinbanding which, as growth progresses, becomes almost undiscernible. No record has been found of an association in tomato of Y virus with tobacco mosaic virus; this is in contrast to frequent references to double virus streak disease caused by joint infection with potato X virus and tobacco mosaic virus.

Infection With Potato Y Virus Alone -- Infections with this virus have been observed throughout the tomato growing season from October to May. In the fall, infections have been few except in small areas within fields. The percentage of infected plants has increased as the season progressed, reaching a peak in late spring when an estimated 70 to 80 percent of the plants in some fields have been affected. Observations suggest that this increase was correlated with the aphid population, which generally increases as the season progresses.

In the field initial symptoms of the disease caused by Y virus alone were the appearance of dark-brown necrotic areas in the laminae of nearly mature leaflets (Fig. 1, A & B). Necrosis usually appeared first and was most severe in the terminal leaflet, but in many cases all leaflets were affected. Usually from two to four adjacent leaves per branch showed necrosis; these leaves were in the late stages of development at the time symptoms appeared. At this stage the youngest leaves showed only faint vein-clearing. Subsequently formed leaves were mildly rugose, slightly distorted, and exhibited an obscure mottle. Leaflets of plants in advanced stages of infection were rolled downward, mostly at the tips, and the petioles also curved downward, giving the plants a drooping appearance. No symptoms have been observed on fruit. Longitudinal purplish striations were found on stems of most varieties but were rarely seen on the Homestead variety. These markings were not necrotic but appeared to result from accumulation of pigments. Affected plants were moderately stunted and with the approach of senescence declined rapidly, especially during hot weather.

Considerable variation in symptomatology, believed attributable to seasonal weather conditions, has been observed. In the fall necrosis seemed to begin in the smallest veins of the leaflets and progressed rapidly until often the entire blade was involved. These withered leaflets frequently remained attached to the petiole for two or three weeks. In the spring necrosis generally appeared as spots, not usually involving the entire blade. On the other hand, the drooping aspect of diseased plants was most pronounced during early fall and late spring, but was not prominent during the cooler days of winter. Similarly, stunting was more severe and plant decline more rapid during warmer days of late spring.

The virus was readily transmitted by mechanical inoculation to Nicotiana glutinosa L. and to N. tabacum L. (var. Havana 38). Vein clearing appeared on both hosts, and rather prominent mosaic ultimately developed. Failure of the virus to infect Datura stramonium L., and its transmission by Myzus persicae Sulz. conform to known properties of the potato Y virus. Since most isolates of Y virus produce on Havana 38 tobacco only slight symptoms, a cross protection test was possible. Tobacco plants systemically infected with a known strain of Y virus remained nearly healthy in appearance after inoculation with the isolate from tomato.

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[1]Florida Agricultural Experiment Station Journal Series, No. 178.

[2]Johnson, E. M. 1930. Virus diseases of tobacco in Kentucky. Kentucky Agr. Exp. Sta. Bull. 306.

[3]Smith, K. M. 1949. Virus diseases of the tomato. Agriculture 56(3): 119-122.

[4]Sakimura, K. 1953. Potato virus Y in Hawaii. Phytopath. 43: 217-218.

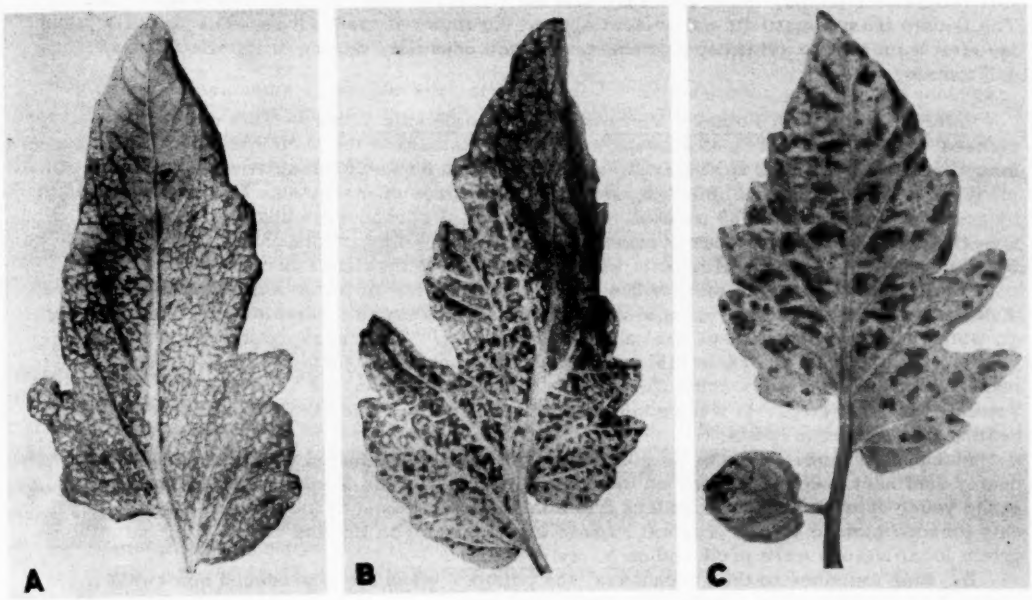

FIGURE 1, A and B. Tomato leaflets showing necrosis seen in the fall on plants naturally infected with potato virus Y. C. Leaflet from plant naturally infected with potato virus Y and tobacco mosaic virus. In spring-crop tomatoes necrosis from Y virus alone was similar to this.

FIGURE 2. Portion of tomato leaf showing symptoms common on plants in advanced stages of infection with potato Y virus and tobacco mosaic virus.

Control tobacco plants, inoculated only with the tomato isolate, developed prominent mosaic. The isolate from tomato did not protect against cucumber mosaic virus. The potato Y virus isolated from tomato evidently represents a strain unusually severe in its effects on tobacco and tomato.

Infection With Both Potato Y Virus And Tobacco Mosaic Virus -- This combination of viruses has been seen in most seasons since 1947, usually in the spring, but has caused severe loss only during the 1952 spring crop, when 3000-4000 acres of tomatoes were affected. In many fields all plants were infected, and yield reductions of marketable fruit were estimated by growers to range up to 50 percent. Two patterns of spread were observed. In one, Y virus appeared to spread in tomatoes previously showing symptoms of tobacco mosaic virus infection. In other instances, spread of the two viruses appeared to be simultaneous.

The combined effect of the Y virus and the tobacco mosaic virus was much more severe than that produced by either virus alone. Plants affected by this complex practically ceased growing, and the production of fruit sharply decreased. The leaves exhibited a pronounced yellow mottle and considerable distortion, and were severely stunted (Fig. 2). The development of necrosis was not much different than that described for the Y virus alone (Fig. 1, C). Fruits from affected plants were somewhat misshapen and lacked the gloss characteristic of healthy mature-green fruits.

Mechanical inoculations to N. glutinosa from tomatoes infected with both viruses produced nearly confluent necrotic lesions on inoculated leaves. Potato Y virus later became systemic in the young upper leaves. Transfers from old necrotic tissue of inoculated leaves yielded only tobacco mosaic virus. Typical mosaic was produced on Havana 38 tobacco, but only necrotic local lesions were produced on N. sylvestris.

In young tomatoes in the greenhouse, the potato Y virus alone produced somewhat more severe mosaic symptoms than did the tobacco mosaic isolate. Both viruses inoculated together to tomato induced markedly more severe symptoms than either virus alone.

Observations in Dade County have suggested that weeds were one of the primary sources of Y virus. Infected tomato plants were most numerous in those parts of fields bordered by citrus and avocado groves but were infrequent alongside pinewoods. Broad-leaved weeds are common in groves, whereas grasses predominate in pinewoods. That weeds were a source of Y virus is also indicated by the fact that infections were found in fall-crop tomatoes several weeks before potatoes were planted in the area.

UNIVERSITY OF FLORIDA SUB-TROPICAL EXPERIMENT STATION, HOMESTEAD, FLORIDA, AND DIVISION OF TOBACCO, MEDICINAL AND SPECIAL CROPS, U. S. DEPARTMENT OF AGRICULTURE

CONTROL OF FIRE BLIGHT OF APPLES IN OHIO IN 1953

H. F. Winter and H. C. Young[1]

During the spring and summer seasons of 1952 and during the winter of 1953, greenhouse tests employing several antibiotics as foliage sprays indicated that streptomycin and terramycin were very effective in preventing fireblight infection on young Jonathan apple trees. In this series of experiments, young rapidly-growing trees were sprayed on from three to five successive days with dosages of 60 to 200 p.p.m. of streptomycin and terramycin. On the day of the last spray application the trees were inoculated with Erwinia amylovora by needle puncture in succulent, fast-growing twigs. Trees sprayed on three or more successive days with 60 p.p.m. or more of either streptomycin or terramycin (commercial grade used) escaped infection when the twigs were inoculated on the last day of spraying. When the sprayed trees were inoculated seven days after the last spraying, 100 percent infection resulted, indicating that the effect of the antibiotic lasted less than seven days.

In the spring of 1953, streptomycin 60 p.p.m. and 120 p.p.m., terramycin 60 p.p.m. and 120 p.p.m., and Dithane Z-78 2 lbs. per 100 gal. were applied to bearing Jonathan apple trees as blossom sprays for the control of blossom and twig blight. Triton B-1956, 2 oz. per 100 gallons of water, was added as a spreader to all of the sprays. The treatments were made on single-tree plots, replicated three times, and randomized. To insure a high incidence of disease, all trees were inoculated in full bloom by spraying them with a bacterial suspension made by adding 10 liters of a three-day culture of Erwinia amylovora in nutrient broth and 10 lbs. of pyrophyllite to 300 gallons of water. The inoculation was made with a high pressure (600 lbs. p.s.i.) power sprayer.

Three applications of the antibiotics were made, the first on May 7, in early bloom; the second on May 9, in full bloom; and the third on May 14, in petal fall. Two applications of Dithane Z-78 were made, one on May 7 and the other on May 9. The inoculation with Erwinia amylovora was made on May 8. Very abundant infection resulted on the control trees, the first blight being seen on May 20, 12 days after inoculation. Blossom and twig counts were made on May 25, 17 days after inoculation. Natural infection during the blooming period was non-existent as was indicated on uninoculated trees nearby in the orchard. The results of this test are given in Table 1.

Table 1. Fireblight control on inoculated orchard apple trees, Jonathan variety, Wooster, Ohio. 1953.

Material[a]	Concentration (p.p.m.)	Percent infection[c]	
		Blossom clusters	Terminal twigs
Untreated	—	83.1	20.6
Dithane Z-78	2400[b]	44.0	9.8
Streptomycin	120	2.0	0.0
	60	7.8	0.5
Terramycin	120	9.0	0.2
	60	20.6	5.2

[a]All trees were sprayed with micronized sulfur up to and including the pink spray.

[b]Equals 2 lbs. in 100 gallons water.

[c]Counts made May 25, 17 days after inoculation. Infection was apparently all the result of inoculation as blight was entirely absent at this time on uninoculated trees.

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Some increase in blight has occurred since the counts were made on May 25. This apparently was a result of late natural infection or spread from infections resulting from the inoculation. As of July 21, however, the streptomycin-sprayed trees were still in excellent condition, showing only a few infections. The control trees, on the other hand, are very severely blighted, with little fruit on them and with many large branches killed. Dithane Z-78, while giving about 50 percent control of both blossom and twig blight early in the season, did not give satisfactory commercial control in this very severe test. Spread of blight was very rapid on this plot later in the season.

While three applications of the antibiotics were made, it is believed that the third one made at petal fall stage was of little effect in this test since it was obvious that nearly all infection resulted from the inoculation made six days earlier on May 8.

Since in the greenhouse work the antibiotics were applied as foliage sprays and since the inoculations were made to the interior of the succulent twigs, it is believed that sufficient quantities of antibiotics were absorbed and translocated to provide temporary immunity to the fireblight bacteria. The action of the antibiotics here at least appeared to be systemic rather than as surface protectants.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OHIO AGRICULTURAL EXPERIMENT STATION, WOOSTER

PROTECTIVE COVER -- A FACTOR INFLUENCING THE DEVELOPMENT OF DWARF BUNT IN WINTER WHEAT

L. J. Tyler and N. F. Jensen[1]

Observations made in fields of winter wheat in New York State in the past nine years, including the current year, show that plants affected by the dwarf bunt fungus (Tilletia brevifaciens G. W. Fischer) often occur in localized areas within a field. Holton et al. (Phytopath. 39: 990. 1949) called attention to the occurrence of this phenomenon in the Pacific Northwestern wheat growing areas which they studied. They obtained some experimental evidence that dwarf bunt was increased in winter wheat by compacting the infested soil immediately after seeding. Experiments were performed by the writers, in 1949-50, in which the incidence of dwarf bunt in rows of wheat mechanically packed after seeding was compared with that in rows not compacted. In the latter experiments there was no evidence that compacting the soil influenced the incidence of dwarf bunt.

Spottiness of dwarf bunt in New York wheat fields suggested to the writers that snow cover and, perhaps, other forms of protective cover on wheat growing on soil infested by the dwarf bunt fungus might influence dwarf bunt development. Accordingly, an experiment designed to test this hypothesis was undertaken at Ithaca, New York, in a wheat planting made in the autumn of 1952. The results obtained in this preliminary test seem to justify their publication at this time.

Hot-water-treated seeds of the variety Cornell 595 were planted 2 to 2 1/2 inches deep in open rows. Seed inoculated with spores was covered with soil free from dwarf bunt spores. Spore-free seed was covered with a mixture of soil and spores (See footnotes, Table 1). The seeding was done October 9, 1952.

Oat straw was applied to one-half of the plots on November 30, 1952. At this time the soil was frozen to a depth of one-half to two inches, soil moisture was high, the wheat plants were 3 1/2 to 6 inches tall and very meagerly stooled. By April 1, 1953 the wheat had resumed very slow but steady growth and on April 9 the straw was removed from the plots.

Snow cover during the winter was not continuous. Plots covered with straw retained the snow longer than did uncovered plots but, because of light snowfall and repeated thawing temperatures during the winter months, all plots were free of snow for at least half of the four-month winter period.

Results included in Table 1 show that 1) straw cover favored development of dwarf bunt, 2) that inoculum applied to the soil was consistently more effective under the prevailing conditions than that applied directly to seed, and 3) that the age of the different collections of inoculum used in the experiment had no effect on the incidence of dwarf bunt.

The writers are not prepared to explain the reasons for the increase in dwarf bunt that resulted from covering plots with straw. Of considerable importance is the fact that stands of wheat in plots covered with straw were much thinner and somewhat later maturing than were those in open plots, thus, the growth of the wheat was definitely retarded by the straw cover. Soil temperature and soil moisture were undoubtedly affected by the straw cover and it may be that these factors had some influence on compactness of the soil. Light at the plant level almost certainly was reduced by the application of straw and this may have had some influence on the physiological processes in the over-wintering wheat.

Studies made by Holton (Phytopath. 33: 732-735. 1943) and by Lowther (Phytopath. 38: 309-310. 1948) show that the process of chlamydospore germination in the dwarf bunt fungus is very sensitive to moisture and temperature. Assuming that straw cover influenced soil moisture and soil temperature it may well be that spore germination and subsequent growth of the fungus were thus indirectly affected.

Results obtained from the preliminary experiment described here show that the protective cover of straw induced, in some way, an increased incidence of dwarf bunt. Repeated observations made in New York wheat fields have revealed that the incidence of dwarf bunt is frequently higher on the leeward side of fence rows, hedge rows, and hilltops. Some New York farmers have reported an increased incidence of dwarf bunt on the leeward side of snow fences set up in their wheat fields compared with that in unprotected parts of their fields. Therefore, two kinds of evidence exist to support the theory that protective cover in some way influences the development of dwarf bunt. The spottiness of dwarf bunt in a given field may well be related to the unequal deposition of snow and/or its persistence in some parts of a field. The apparent cyclic development of dwarf bunt in some Pacific Northwest regions, suggested by some workers, may be partly related to the amount, distribution, and persistence of snow cover.

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Table 1. Development of dwarf bunt in wheat on plots covered with straw compared with uncovered plots. Ithaca, New York; plots planted autumn 1952.

Inoculation method	Year inoculum collected	Percentage heads infected by dwarf bunt[a]			
		Plots			
		Covered[b]	Open[c]	Covered	Open
Spores[d] applied to seed	1950	4.3	0.6	5.4	0.0
	1951	5.2	0.0	4.7	0.0
	1952	4.2	0.0	3.5	2.6
Spores[e] applied to soil	1949 & 1950 mixed				
	1951	6.1	0.0	20.1	0.0
	1952	15.3	1.4	17.2	6.3
		13.0	2.5	6.7	1.8

[a]Percentage of total heads in 4-row plots, each row 5 feet long; duplicate plots.
[b]Covered = Oat straw applied loosely to depth of 2 to 3 inches.
[c]Open = Plots were not covered, wheat exposed to prevailing environment.
[d]Spores powdered and dusted on seed at rate of 1/4 gm. per 100 gm. seed.
[e]About 1/4 gm. of spores diluted with talc was mixed with soil necessary to cover seed in a 5-foot row.

The writers expect to continue their studies in an effort to answer some of the questions posed above. It is hoped that use can be made of protective cover, such as straw, in testing wheat lines for their reaction to the dwarf bunt fungus and in testing seed treatment chemicals.

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RELATIVE PREVALENCE AND SEVERITY OF THE DISEASES OF FORAGE LEGUMES IN WISCONSIN, 1946-1952

Earle W. Hanson

The observations reported here are based on regular State-wide surveys made in June and August of each year, together with field inspections at other times as opportunities occurred.

Weather Conditions

The prevailing weather conditions for Wisconsin for the growing seasons (April 1 to October 30) of the years covered by this survey were determined from the records of the U. S. Weather Bureau. While considerable differences in temperature and precipitation sometimes occurred from one locality to another, the averages for the State were as follows: 1946 was cooler than normal, and much drier except during June and September. The first four months of 1947 were colder and the last three months were warmer than normal. Precipitation was above normal the first three months, about 80 percent of normal in July, slightly above normal in August, normal in September, and below normal in October. 1948 was warmer and much drier than normal. There was a deficiency in precipitation each month from May through October. The effect of this drought on stands and vigor of legumes was very adverse. More than half of the clover seedings made in the spring of 1948 were failures. Most second-year fields of red clover also had poor stands and gave poor forage yields, especially in the second hay crop. The winter of 1947-48 was more unfavorable than usual and caused much injury to legumes, especially red clover. 1949 averaged a little warmer and drier than normal, although precipitation was above normal in both June and July. 1950 was considerably cooler and wetter than normal. 1951 was cooler and wetter than normal. In 1952, temperatures averaged above normal for the period of April through September, but October was considerably colder than normal. April was dry and September and October were very dry; precipitation was adequate in most areas during the other months.

Diseases of Red Clover

Northern anthracnose (Kabatiella caulivora) was of minor economic importance during this period. It was present every year but usually only in trace amounts. In 1946, 1948, 1949, 1950, and 1951, it was of no consequence. In 1947 it was generally prevalent over most of the State and caused some damage. The epidemic developed late so that the first hay crop was cut and removed before it was greatly injured. However, the second crop also became infected, the severity ranged from light to moderate, and there was a small amount of damage in a large number of fields. New seedings showed only a trace of infection. In 1952, the disease was found only in a few scattered locations but some damage occurred in the extreme northern part of the State.

Black stem (Phoma trifolii) was present in every field every year and caused some damage in most years. It was not severe during the first half of the growing season so it had little effect on the first crop of hay. It was most abundant and severe in the fall, particularly when the weather was cool and wet. The characteristic stem blackening increased when cutting of the clover was delayed, hence it was most severe in fields left for seed. There seemed to be a high positive correlation between maturity of the stems and the amount of blackening. Severity ranged from light to severe.

In the fall of 1946, the disease was generally prevalent and moderately severe. In 1948 it was very light and of no importance. In 1947, 1949, 1950, and 1951, the severity ranged from moderate to severe and considerable damage resulted in fields left for seed as well as in fields cut late for hay. In 1952, it was much less severe than in 1951 and was abundant only in fields left for seed where it did cause some blackening of stems and petioles. During this survey the disease was not important on new seedings.

Powdery mildew (Erysiphe polygoni) was of secondary importance during these years. It was present in every field every year but did not become abundant until fall. There never was more than a trace on the first crop of hay and the amount on the second cutting ranged from a trace to light. Most of the mildew developed after the first week in August on the aftermath, on new seedings, and on fields left for seed. During the months of September and October, it was moderately severe in 1946, 1947, 1948, and 1949 and light in 1950, 1951, and 1952.

Rust (Uromyces trifolii fallens) was present every year but was of no consequence on the first crop of hay. The severity in second crop stands, fields left for seed, and new seedings ranged from a trace to severe. Rust was very light in 1946, being most prevalent in the western part of the State. In 1947, the severity ranged from a trace to moderate but there was little damage. In 1948 and 1949, most fields had only light infection. In 1950, the disease was generally prevalent and abundant late in the season, particularly on new seedings, but did not appear to cause much damage. In 1951, it was abundant and severe on the leaves and stems of second-year stands and unusually abundant on the leaves of new seedings. In the fall of 1952, rust was again abundant on the leaves of new seedings and was severe on second-year stands left for seed, particularly in the extreme northern part of the State where it not only killed many of the leaves but also produced large pustules on the stems which accentuated the effects of the drought and caused premature browning of the plants.

Stemphylium leaf spot (Stemphylium sarcinaeforme) is one of the most common leaf spots of red clover in Wisconsin. It was present in all fields every year. It was most prevalent and severe during the latter part of the summer and fall. In 1946, its severity ranged from light in most parts of the State to moderate in some fields in the northern half of the State. In 1947, it was generally prevalent and moderately severe in most fields. In 1948, the disease was very light. In both 1949 and 1950 it was abundant, moderately severe, and caused considerable damage. In 1951, the severity ranged from light to moderate. In 1952, it was light in all parts of the State except the extreme northern part where it was moderately severe. While this disease was not of major importance during the years of this survey it was the most destructive of the leaf spot diseases of red clover.

Bacterial leaf spot (Pseudomonas syringae) was present in greater than trace amounts only in 1947, 1949, and 1950. In 1947 it was quite general in distribution but light in severity. In 1949, it was more prevalent in the southern part of the State than elsewhere and was moderately abundant during June and July. In 1950 it was generally prevalent and moderately severe, but did not cause important damage.

Cercospora leaf and stem spot (Cercospora zebrina) was found in trace amounts from 1946 to 1951, inclusive. In 1952, it was the most common leaf spot on red clover but did not cause important damage. Infection occurs at any time during the growing season on plants of any age but the disease is usually most abundant during late summer and fall. In 1952, however, it was most abundant during June and July.

Pseudopeziza leaf spot (Pseudopeziza trifolii) was of no economic consequence during this period. It was found only in trace amounts in 1946, 1948, 1949, 1950, and 1951. In 1947 and 1952 it was generally prevalent; the severity was light in 1947 and ranged from light to moderate in 1952.

Sooty blotch (Cymadothea trifolii) is usually of minor importance on red clover in Wisconsin. In 1946, 1947, 1948, and 1949, it was of no consequence. In 1946, it was present in moderate amounts late in the fall in two counties of west central Wisconsin; elsewhere there was only a trace of infection. In 1947, a few fields showed moderate to severe infection, but most fields had only a trace. Sooty blotch was virtually absent in 1948. In 1949, it was present in trace to light amounts in northeastern and east central Wisconsin. In 1950, it was generally prevalent and more abundant than usual. It reached epidemic proportions and caused some defoliation in a considerable number of fields in the extreme northern part of the State. In 1951, it was present in most fields but again was most abundant in the northern half of the State. In 1952, it was of consequence only in the extreme northern part of the State where it was moderately severe.

Blackpatch (Unidentified fungus) was of no consequence during this period. It was not found in 1946 or 1948 and in the other years it was present only in trace amounts of a few scattered plants.

Virus diseases (Complex[1]) were prevalent in every field every year. The viruses isolated from field stands of red clover during this survey included: the red clover vein-mosaic virus, the pea common mosaic virus, the bean yellow mosaic virus, the alsike clover mosaic virus, the Wisconsin pea streak virus, and the alfalfa mosaic virus. In addition, several others were obtained which have not been identified. The red clover vein-mosaic virus was the most prevalent during this period. The importance of these diseases has not been ascertained.

The average prevalence of the virus complex in 1946 ranged from a trace in most fields to moderate in others. In 1947, the incidence was light. In 1948, virus diseases were unusually

[1]Hanson, E. W., and D. J. Hagedorn, 1952. Red clover, a reservoir of legume viruses in Wisconsin. (Abstr.) Phytopath. 42:467.

prevalent. This was probably due in part at least to the presence in most clover fields of large populations of aphids and leaf hoppers during the early part of the summer. In 1949, 1950, 1951, and 1952, virus diseases were generally prevalent in moderate amounts.

Root and crown rots (a complex[2]) constituted the most important group of diseases during this period. They were present every year in light to abundant amounts and caused some damage every year. Their effects were usually confounded with the effects of other factors so that it was difficult to ascertain the damage due to the rots alone. These diseases were particularly severe in areas and years where there had been winter injury, insect infestations, drought, or other predisposing factors.

In 1946, stands were poor and short due to drought, and root and crown rots were generally prevalent and moderately severe in most parts of the State. The clover root borer (Hylastinus obscurus) was abundant and caused severe damage to second-year stands in east central Wisconsin. Crowns and roots of infested plants were completely rotted so that by September 1 more than 90 percent of the plants in some fields were dead. New seedings were not injured appreciably.

In 1947, most fields yielded a good first crop of hay but subsequently many of them failed to recover and large percentages of plants died. Some fields in south central Wisconsin were plowed after the first hay crop had been harvested because of poor recovery. Roots and crowns of the plants in such fields were severely rotted. There were also reductions in stand of new seedings, particularly during the hot part of the summer. The clover root borer was present in much of eastern Wisconsin but was not as abundant as in 1946.

The winter of 1947-48 began suddenly in November before the plants were properly conditioned and was unusually severe. The result was a marked reduction in stand. Many fields were plowed in the spring when they failed to resume growth. Most of the remaining fields had only fair stands and the surviving plants were mostly injured, weak, and ready prey for root and crown rots which further reduced stands. The summer of 1948 was extremely dry and unfavorable for the development of red clover. The clover root borer was abundant in east central Wisconsin. Root and crown rots were severe.

In 1949, second-year stands were mostly poor to fair as a result of the unfavorable conditions in 1948. Rots were present in all fields. Severity ranged from light to moderate by June and moderate to severe by September. The rots caused some damage but not nearly so much as in 1948.

In the early spring of 1950, there was severe winter injury to all legumes in many parts of the State. Red clover was injured more than alfalfa or sweetclover. Most injury occurred in eastern, central, and southern parts of the State due to lack of snow cover. In the western and northern parts of Wisconsin where there was good snow cover there was little injury. In some localities where injury was especially severe, a large majority of the fields were plowed in the spring and the remaining fields had only fair stands. Root and crown rots were abundantly present on injured plants and there was severe stunting in some fields. The rots were not severe in new seedings.

All legumes came through the winter of 1950-51 in excellent condition. The soil remained unfrozen most of the winter due to heavy snow cover. New seedings also produced excellent stands and appeared to be in very good condition as of October 1951. Root and crown rots were of minor importance in 1951. They were more prevalent and severe on second-year than on first-year plants but were not so destructive as in many other years.

The winter of 1951-52 was not quite so favorable as the preceding winter but, with the exception of a few unadapted strains being tested in the State, red clover came through the winter in excellent condition. Root and crown rots were less severe than usual.

Botrytis blight (apparently a Botrytis of the cinerea type[3]) was found on red clover blossoms in the late summer and fall of 1951 and again at about the same time in 1952. It was of no importance on red clover. It was more abundant on alfalfa and sweetclover. Red clover grown in mixtures with alfalfa had more blight than pure stands of red clover.

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[2]Kilpatrick, R. A., and E. W. Hanson. 1951. Relative prevalence of fungi associated with root and crown rots of red clover in Wisconsin. (Abstr.) Phytopath. 41:21.

[3]Jones, F. R., 1952. Botrytis on flowers of alfalfa, sweetclover and red clover. Plant Dis. Reptr. 36:61

Diseases of Ladino Clover

Pepper spot (Pseudoplea trifolii) was probably the most common and widely distributed leaf disease during this period. It was found in every field every year. However, in most cases it was abundant only on the lower leaves and did not cause much damage.

Cercospora leaf spot (Cercospora zebrina) was found in trace to light amounts from 1946 to 1951. In 1952, it was very abundant and caused some damage in many fields. It was generally prevalent but most severe in the southern part of the State.

Sooty blotch (Cymadothea trifolii) was found every year in most fields but was present only in trace to light amounts from 1946 to 1949. In 1950, 1951, and 1952 it was more abundant, particularly in the northern part of the State. It was of little economic importance.

Stagonospora leaf spot (Stagonospora meliloti) was common during periods of cool, wet weather but was of minor importance. The severity ranged from a trace to moderate.

Pseudopeziza leaf spot (Pseudopeziza trifolii) was of no importance. It was found only in trace amounts except in 1947 and 1952 when it was somewhat more abundant.

Rust (Uromyces trifolii trifolii-repentis) was observed only in trace amounts.

Virus diseases (Complex) were prevalent in every field every year and were moderately severe most years. Infected plants showed a great variety of symptoms suggesting the presence of at least several viruses. Little was done to identify the viruses occurring in nature on this crop, but enough isolations and inoculations were made to ascertain that viruses were present. In greenhouse tests it was established that several of the viruses occurring naturally on red clover in Wisconsin could infect ladino clover.

Root and stolon rots (Complex) were present every year and ranked among the most important diseases of this crop. As in the case of red clover, the severity of the rots was closely associated with the amount of injury during winter and early spring. Stolon rots were most severe in those years and localities in which the plants lacked snow cover and the stolons were first injured by unfavorable temperatures, or ice. Rhizoctonia, Fusarium, and several other fungi were isolated from the margins of lesions of diseased plants. However, when the isolates were used to inoculate the roots and stolons of healthy, vigorously growing plants in the greenhouse little or no disease developed. Apparently many of the fungi associated with these rots are weak pathogens and cause severe injury only under certain environmental conditions or when the plants are first predisposed by other factors. The problem of the nature and causes of this group of diseases needs further investigation. Sclerotinia trifoliorum was never isolated during this survey.

Diseases of Alfalfa

Bacterial wilt (Corynebacterium insidiosum) has been a major disease of alfalfa in Wisconsin. It is widely distributed but has been most important in the southern and central parts of the State and least important in the northeastern part. Winter injury predisposes plants to infection and the amount of disease usually increases following such injury. With a few exceptions, wilt was important only in the older stands. Losses due to this disease are on the decline due to the current practice of using shorter rotations and the growing of strains of alfalfa resistant to wilt.

Spring black stem (Ascochyta imperfecta) was generally prevalent and moderately severe in May and June of 1947, 1950, and 1951 when the weather was cooler and wetter than normal. It was present but of less importance in the other years.

Summer black stem (Cercospora zebrina and Ascochyta imperfecta) was found in every field every year but was not of major importance. It was very light in severity in 1946 and 1948. In the other years, the severity ranged from light to moderate. In 1951, a few fields showed very extensive blackening of the stems, petioles, and even the rachises. In 1952, there was considerable leaf spot but not so much blackening of the stems as in some years.

Common leaf spot (Pseudopeziza medicaginis) was the most prevalent and widely distributed leaf spot on alfalfa. It occurred every year, caused some losses in foliage most years, but was not of great economic importance. Its severity was very light in 1946. In 1947, it ranged from light to moderate. In 1948, there was only a trace of the disease on the first crop of hay; on the second crop, the severity ranged from a trace to moderate with only about 40 percent of the fields having more than a trace. In June of 1949, the severity ranged from light to moderate and was mostly limited to the lower 50 percent of the leaves. On August 23, it was severe in southwestern Wisconsin and moderately severe elsewhere. In 1950 there was only a trace of the disease during the first part of the summer. On the second hay crop, the severity ranged from

light to moderate. It was most severe in southern Wisconsin and least severe in central Wisconsin. In 1951, the disease was very abundant and moderately severe over the entire State. In 1952, it occurred in light amounts on the first hay crop but increased so that most fields had moderate amounts of the disease by late fall. During the years of this survey, new seedings of alfalfa showed only a little leaf spot during the first part of the summer. By late fall the disease was often moderately abundant but seemed to have little adverse effect on the plants.

Yellow leaf blotch (Pseudopeziza jonesii) was present every year but was of little importance except in 1950 when it was very severe. By June of that year, it was prevalent and severe over the entire State. It destroyed at least 20 percent of the leaves of the first hay crop and in some fields the loss of leaves exceeded 50 percent. The disease was also generally prevalent on the second crop but was less severe in most areas. In central Wisconsin, particularly in the Marshfield area, it was very severe throughout the growing season; even the top leaves had abundant lesions. In the other years it was not nearly so generally prevalent or abundant as the common leaf spot.

Stemphylium leaf spot (Stemphylium botryosum) was present throughout the State in trace to light amounts but was of no consequence. It could scarcely be found during the dry summers of 1946 and 1948. In the other years it was present in nearly every field, being slightly more abundant in 1949, 1950, and 1951.

Stagonospora leaf spot (Stagonospora meliloti) was common during periods of cool, wet weather but was of little consequence. The severity ranged from trace to light.

Cercospora leaf spot (Cercospora zebrina) was observed in trace amounts from 1946 to 1951. In 1952, it was generally prevalent and more abundant than usual but caused little damage.

Downy mildew (Peronospora trifoliorum) was observed during the first half of the summer of each year except 1946 and 1948. It was widely distributed but of little importance. Only in 1950, in a few fields in southcentral Wisconsin, was there more than a trace of this disease.

Rust (Uromyces striatus medicaginis) was found in occasional fields in trace to light amounts throughout the State every year except 1946 and 1948. It was of no importance.

Virus diseases (alfalfa mosaic virus and probably others) were commonly present and widely distributed. The kinds of alfalfa viruses occurring in the State were not determined but alfalfa mosaic virus was isolated from red clover. The importance of these diseases was not ascertained but probably they were of minor consequence during the years of this survey.

Root and crown rots (Complex) comprised one of the most important group of diseases. Their effects were usually confounded with the effects of winter injury, bacterial wilt, and other factors so it was impossible to determine the damage caused exclusively by the rots. The severity of this disease complex ranged from light to severe with the greatest damage occurring in 1948 and 1950. There was also severe injury to old stands in 1952. See discussion of root and crown rots of red clover.

Botrytis blight (apparently same as on red clover[3]) was found abundantly on the inflorescences in the late summer and fall of 1951 and 1952. It had little effect on forage production but virtually prevented seed formation in the infected flowers. The fungus grew over the entire inflorescence, covering it with its coarse mycelium, and causing the florets to turn brown but not to drop off readily. Since no inoculation experiments were made, it was not definitely determined whether the Botrytis incited the blight or was secondary but it certainly was abundantly associated with the disease.

Diseases of Sweetclover

Black stem (Mycosphaerella lethalis, Cercospora davisii, Ascochyta imperfecta, and possibly others) was the most common, conspicuous, and important disease of sweetclover in Wisconsin during this period. It was present in every field every year. The severity ranged from a trace to severe with most fields showing light to moderate infection by late summer. Spring black stem was generally prevalent during the cold wet conditions of May and June of 1947, 1950, and 1951. It was present only in trace amounts in the other years. Summer black stem was very abundant in 1947, 1949, 1950, and 1951 and less severe in the other years. It was most abundant during the latter part of the summer and fall. There was considerable overlapping of the spring and summer black stem diseases and it was not always easy to distinguish between them on the basis of symptoms.

Stem canker (Ascochyta caulicola) was of little consequence. It was observed on a few plants every year in an occasional second year stand. It was most severe in experimental nurseries where it sometimes caused extensive cankers and malformations in certain stocks.

Phytophthora root rot (Phytophthora cactorum) was found in trace amounts every year except 1946 and 1948 but was of no economic importance. It was somewhat more prevalent in 1947 than in the other years.

Miscellaneous root rots (Complex), other than Phytophthora root rot, were present every year and were important in the years of severe winter injury.

Stagonospora leaf spot (Stagonospora meliloti) was common during periods of cool wet weather but was not important. The severity ranged from trace to light.

Cercospora leaf spot (Cercospora davisii) was found in trace amounts each year. In 1952, it was generally prevalent and more abundant than usual but caused little damage.

Pseudopeziza leaf spot Pseudopeziza meliloti) was rarely observed on sweetclover and was of no consequence. It was present in trace amounts in 1952.

Virus diseases (Complex) were present in all fields every year in light to moderate amounts. No attempt was made to determine the relative prevalence of the various viruses present in commercial fields or to ascertain their importance. It was determined by greenhouse tests that several of the viruses common in Wisconsin fields of red clover and other legumes readily infect sweetclover.

Botrytis blight (apparently same as on alfalfa and red clover[3]) was present on the inflorescences of sweetclover in the late summer and fall of 1951 and again at about the same time in 1952. It was of no economic importance.

DIVISION OF FORAGE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, AND WISCONSIN AGRICULTURAL EXPERIMENT STATION

OBSERVATIONS ON BLACK ROOT ROT SYMPTOMS ON COTTON SEEDLINGS AT DIFFERENT SOIL TEMPERATURES[1]

L. M. Blank, P. J. Leyendecker, Jr., and R. M. Nakayama

The first report concerning black root rot on cotton seedlings, caused by Thielaviopsis basicola (Berk. & Br.) Ferraris, was by Sherbakoff (6) in 1940, although this fungus is known to have a wide range of host plants and has been studied extensively on tobacco (1). Sherbakoff reported seedling infection of American-Upland cotton in the greenhouse, in field soil taken from a Fusarium-nematode-infested field in Tennessee. No mention was made of seedling symptoms other than a rotting away of the primary root. In 1942, King and Presley (2) described seedling symptoms of black root rot of American-Egyptian and Upland cotton under greenhouse and field conditions. No observations were reported concerning the effect of soil temperature upon the expression of disease symptoms in the seedling stage. In 1945 and 1946, Presley (5) observed seedling damage and reduction in American Upland cotton stands by Thielaviopsis in Mississippi and estimated that in certain localized areas 20 percent of the seedlings were killed by this fungus. His report establishes definite evidence of seedling infection and damage of American Upland cotton by T. basicola under field conditions.

The discovery of black root rot on mature American-Egyptian cotton plants in Texas and New Mexico in 1951 (3) and further observations on its prevalence in mid-to-late season in 1952 (4) led to the present consideration of the disease on younger plants in the greenhouse. Since nematodes were consistently found on plants showing symptoms of black root rot in the above areas, the following observations on seedlings grown in the greenhouse are presented to show disease symptoms in the presence and absence of nematodes.

PROCEDURE: Two-gallon crocks containing naturally- or artificially-infested steam-sterilized field soil (Anthony sandy loam) were prepared. Forty grams of two-week old cultures of a local isolate of T. basicola, grown on a soil-corn meal medium, were incorporated into the crocks of steamed soil, with an appropriate number as checks. Two weeks later, seed of American-Egyptian cotton, variety Pima 32, was planted in all crocks of soil. Prior to germination of the seed, the crocks containing the different soil treatments were divided and placed in greenhouse units of different mean temperatures for the duration of the experiment. Average soil temperatures for the 70-day experiment were 79°, 77°, and 69° F for the three greenhouse units.

RESULTS: The naturally-infested soil, obtained from a commercial field near Fairacres, New Mexico, initially contained nematodes, and the seedlings grown in such soil developed a limited amount of galling. However, under greenhouse conditions, no difference in the initial occurrence or final severity of black root rot expression could be detected between the naturally-infested soil containing nematodes and the steamed artificially-infested soil.

Throughout the experimental period, disease symptoms in the 77° greenhouse unit were intermediate between those produced in the 79° and 69° units. Therefore, this discussion of black root rot symptoms will be based only upon those observed under the highest and lowest soil temperature conditions.

For clarity, development of disease symptoms in the greenhouse seedlings will be presented as above- and below-ground manifestations.

Above-ground Symptoms -- The first evidence of above-ground symptoms occurred approximately four or five weeks after planting, when the plants were in the 3-4 leaf stage. At this time, stunting of infected seedlings was observed at all soil temperatures, with the most severe reaction occurring at the lowest temperature. Death of the young seedlings had not occurred prior to this time, although 35 days after planting the first evidence of complete collapse of seedlings was observed in the cool temperature unit (See Figure 1, D). Seedling collapse continued to occur in the cool unit, and by 70 days all seedlings in the artificially- and naturally-infested soil were dead. In contrast, only one seedling in the warm unit was killed by Thielaviopsis, although stunting was common, as shown in Figure 1, A.

Under cool conditions, the infected seedlings remained in the stunted condition for some time. This initial symptom was followed by a slight yellowing and wilting of one or more of the leaves, which progressed rapidly to a complete collapse of all leaves in two or three days.

[1]Contribution No. 83 from the New Mexico Agricultural Experiment Station, and the U. S. Bureau of Plant Industry, Soils, and Agricultural Engineering, Division of Cotton and Other Fiber Crops and Diseases.

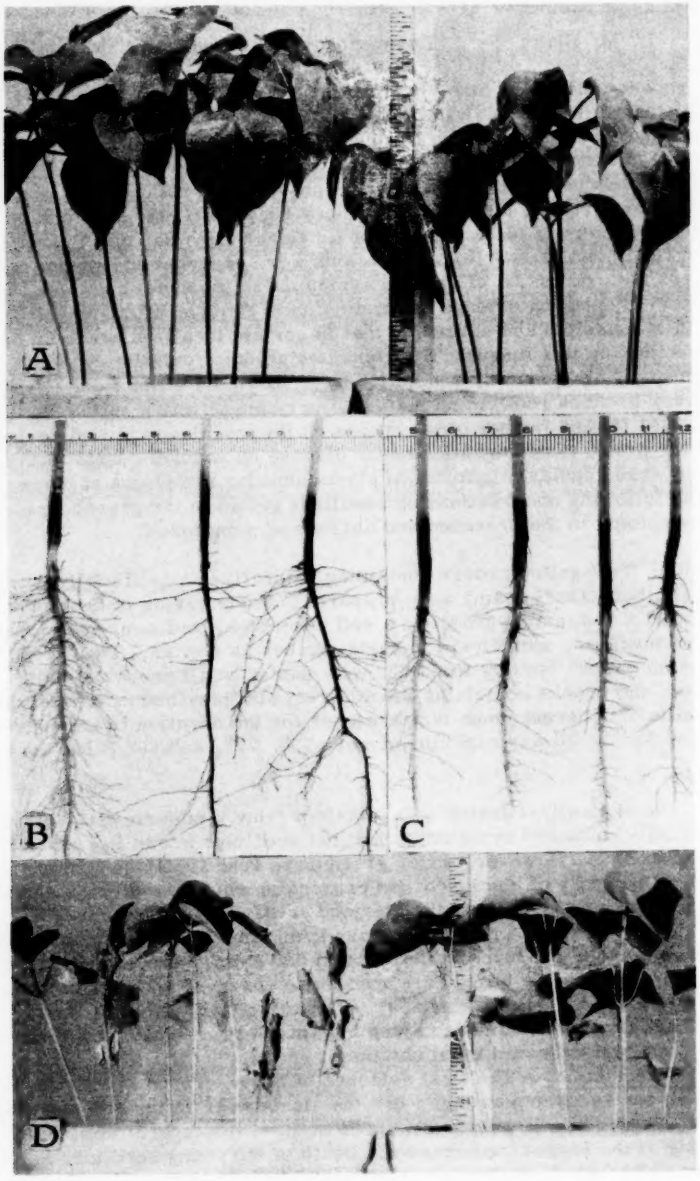

FIGURE 1. Black root rot (Thielaviopsis basicola) on 40-day-old American-Egyptian cotton seedlings. (A) Appearance of seedlings grown in warm house steamed (check) soil, left, and in naturally infested soil, right. (B) Infected roots from artificially-infested soil in cool house. Root on left is healthy. (C) Roots from naturally-infested soil in warm house. Note the infected crown area and occasional lesion on lower tap root. (D) Seedlings from cool house in artificially-infested soil, left, and steamed (check) soil, right.

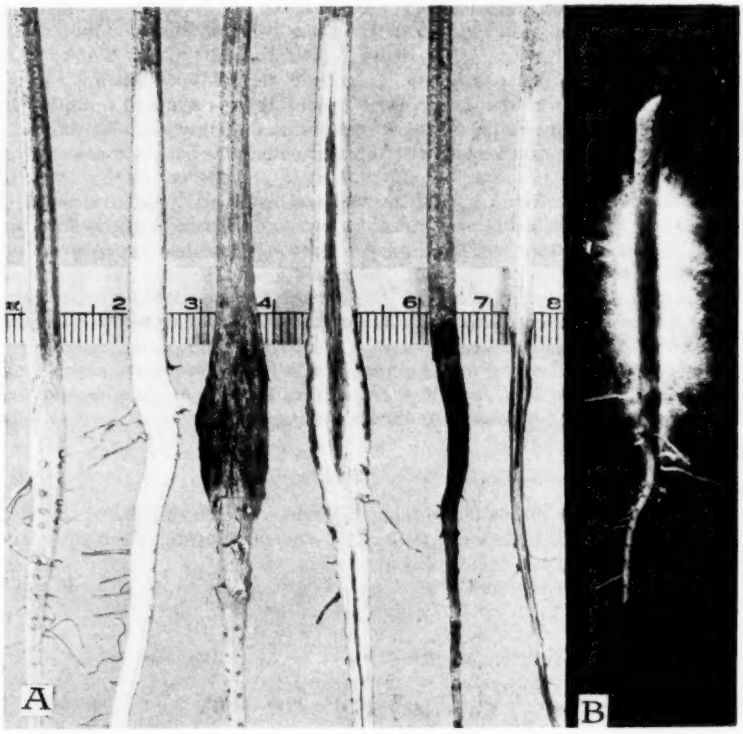

FIGURE 2. Seventy-day-old American-Egyptian cotton roots showing symptoms of black root rot. (A) The two roots at left are healthy checks. The center two show external and internal appearance of roots from the warm house. The two roots at the right are from the cool house. (B) Appearance of infected root-crown after floating in water for three days. The mycelial mass is permeated with characteristic chlamydospores of Thielaviopsis.

Within a week, the above-ground portion of the plant was dead, with the dried leaves remaining attached, as shown in Figure 1, D.

Below-ground Symptoms -- Forty days after planting, representative seedlings in the cool and warm units were washed from the soil. Their condition at this time is illustrated in Figure 1, B and 1, C. The seedlings from the cool unit showed the characteristic blackened root system of Thielaviopsis-infected plants. This discoloration and rotting of the main root occurred near the ground line and extended the length of the root. The infected central tissue was brownish-black in color. When observed under the microscope, the characteristic Thielaviopsis chlamydospores were noted.

At higher soil temperatures, infection and expression of symptoms typically occurred mainly at the crown or just below the soil surface, as shown in Figure 1, C. However, lesions were found occasionally on the lower portion of the tap root. In either case, the extent of the infected tissue and severity of damage was noticeably less than that observed on plants grown under cool soil conditions. The characteristic symptom of Thielaviopsis infection at high soil temperatures was the pronounced swelling of the crown, accompanied by a dark-brown discoloration of the central woody cylinder, as shown in Figure 2, A. It is assumed that, at high soil temperatures, the initial crown infection becomes overgrown with healthy tissue.

With the termination of the greenhouse experiment at 70 days, it was not possible to follow symptom development into mature plants. However, observation of symptoms on field-grown American-Egyptian plants in mid-to-late season in 1951 and 1952 indicates that swelling of the crown frequently occurs. Apparently, under field conditions, the pathogen may remain quiescent

in the infected area of the crown until the soil temperature becomes favorable for renewed activity of the fungus, at which time typical above-ground symptoms develop.

During the course of this investigation, it was found that suspected black root rot of seedlings can readily be determined by placing the tissues in tap water. When T. basicola was present, it grew very rapidly into the water after two or three days and completely surrounded the infected tissue with a fine mycelial network, as shown in Figure 2, B. Under low-power magnification, chlamydospores were seen scattered through the fungal mass.

SUMMARY: The absence of nematodes, in steamed artificially-infested soil, or their presence, in naturally-infested field soil, did not alter the pathogenicity of Thielaviopsis basicola on American-Egyptian cotton seedlings grown at several soil temperatures in the greenhouse.

With average soil temperatures of 69°, 77°, and 79° F. the disease expression was most severe at the lowest temperature. This is in accord with the findings of other workers studying black root rot on tobacco.

At higher temperatures, stunting of the plant was the characteristic above-ground symptom. Usually, the infection remained localized in the crown area, and a pronounced swelling developed. Death of the infected plants and necrosis seldom occurred.

Literature Cited

1. Dickson, J. G. 1947. Diseases of Field Crops. McGraw Hill Co., New York.
2. King, C. J., and J. T. Presley. 1942. A root rot of cotton caused by Thielaviopsis basicola. Phytopath. 32: 752-761.
3. Leyendecker, P. J., Jr. 1952. Root rot of cotton caused by Thielaviopsis basicola discovered in the Upper Rio Grande Valley of Texas. Plant Dis. Reptr. 36: 53.
4. Leyendecker, P. J., Jr., L. M. Blank, and R. M. Nakayama. 1953. Further observations on black root rot of cotton in the Upper Rio Grande Valley of Texas and New Mexico. Plant Dis. Reptr. 37: 130-133.
5. Presley, J. T. 1947. Thielaviopsis root rot of cotton in Mississippi. Plant Dis. Reptr. 31: 152.
6. Sherbakoff, C. D. 1940. An occurrence on cotton of black root rot caused by Thielaviopsis basicola. Proc. Cotton Disease Council. 5: 4.

NEW MEXICO AGRICULTURAL EXPERIMENT STATION, DEPARTMENT OF BIOLOGY, STATE COLLEGE, NEW MEXICO, AND THE DIVISION OF COTTON AND OTHER FIBER CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE

A BACTERIAL LEAF SPOT DISEASE OF CASTORBEAN NEW FOR THE UNITED STATES

O. H. Calvert, C. A. Thomas, and D. L. Van Horn[1]

A bacterial leaf spot disease of castorbean, Ricinus communis L., apparently not previously reported as occurring in the United States, has been observed on castorbeans in commercial fields in Oklahoma and Texas in each of the years 1950, 1951, and 1952. The damage to certain varieties in these years was slight to moderate. The disease probably occurs throughout the castorbean producing area of the southern Great Plains of the United States. This disease was noted also in varietal yield tests conducted in Maryland and Texas in 1952. The causal organism was isolated, proved to be pathogenic to castorbeans, and tentatively identified as Xanthomonas ricinicola (Elliott) Dowson.

The geographical distribution of this disease appears to be world wide. It has been reported as occurring in Korea, Japan, Russia, Italian Somaliland, and Uganda (2). Patel et al. (3) recently have reported the disease as serious in India. Certain plant introductions received from Paraguay, Nicaragua, Kenya and India appear to have become diseased from seed-borne inoculum, which indicates that the disease probably is present in these countries.

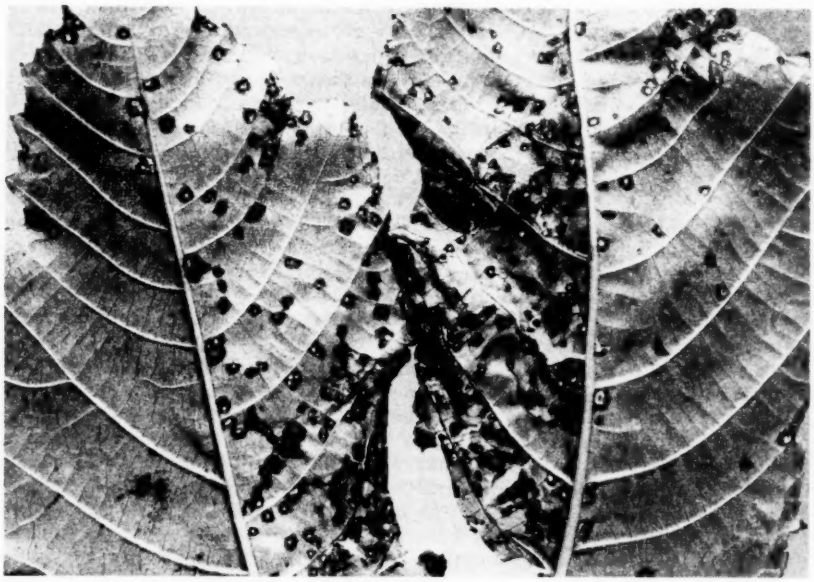

FIGURE 1. Close up of a naturally infected castorbean leaf showing angular and coalesced lesions.

In the field the disease may first be found on the cotyledons of young plants and is first evident as watersoaked lesions that gradually turn black with the drying of the cotyledons. The small, round, water-soaked spots on the young expanding leaves enlarge to 2 to 5 mm. in diameter and may become somewhat angular by being delimited by the veins. Frequently the lesions coalesce and affected leaves gradually yellow and drop prematurely (Fig. 1). Black lesions may occur also on the petioles, stem, and branches and may invade the flowering spikes, causing blasting of the flowers in highly susceptible varieties. Bacterial exudate, which later dries, may be found on the surface of the lesions during humid weather.

Artificial inoculations in the greenhouse caused most damage on young plants. The line

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Nebraska 145-4, which is used as the female parent for hybrid combinations (1, 4), was the most susceptible variety tested. Nebraska 145-4 plants, six days after inoculation in the greenhouse, exhibited visible symptoms in the form of numerous, circular, water-soaked spots less than 1 mm. in diameter. In young leaves the spots enlarged, became angular, and coalesced, producing large necrotic areas which often involved the entire leaf. Young unexpanded leaves appeared to be more susceptible than old leaves. The commercial variety, Cimarron, proved to be quite resistant.

The organism can be isolated easily on nutrient agar. The colonies are at first glistening yellow but later, within four or five days, turn brown, imparting a brown color to the medium. The growth of the bacteria is rapid and a gum-like material is produced in abundance. Since the bacteria soon die on a non-dextrose medium, transfers were made to potato-dextrose agar for storage. Microscopically the bacteria occur in short chains and the measurements of the cells check with those given by Elliott (3), namely, 0.4 to 0.9 X 1.3 to 2.6 microns. Hanging drop preparations of the bacteria indicate them to be flagellate. However, efforts to stain the flagella following several flagella staining methods were unsuccessful.

The organism, from field observations, appears to be seed-borne and to be spread chiefly by wind-blown rain. At Beltsville, Maryland, in 1952, the bacterial leaf spot developed first on the variety Nebraska 145-4 and then spread by rain to the other varieties. The disease developed in the same way at College Station, Texas; i.e., first on one or two varieties (USDA 9/19 and USDA 101) and then spread to other varieties in the test. In early July of 1952, severe symptoms were found in a seed increase field of Nebraska 145-4 at Jones, Oklahoma. The disease first occurred on plants in a poorly drained area following an old watercourse in the field. Shortly after the symptoms were noted, a heavy rain accompanied by high winds occurred, and the disease spread throughout the field and developed rapidly during seven to ten days of humid weather following the rain. At the end of this period many of the first racemes were so badly affected that all the seed were lost. Following the period of high humidity the weather became hot and dry and the disease did not spread to the new growth. While a few plants were killed (those on which the disease affected all the growing points), most of the plants "recovered" completely and produced a good crop of seed on secondary racemes.

At the time the above observations were made at Jones, Oklahoma, on Nebraska 145-4, nearby fields of Conner, USDA 74, and Cimarron varieties were checked for symptoms of bacterial leaf spot. Lesions were quite general on the leaves of Conner and USDA 74, but not severe enough to cause loss of seed by invasion of the racemes. Cimarron appeared to be almost completely resistant.

Some additional field notes on varietal susceptibility under conditions of natural spread were taken at Beltsville, Maryland, and College Station, Texas, in 1952. The relative ratings given each variety may not indicate their true resistance because the disease did not develop uniformly over all the replications at either location. No variety was found to be immune, and none so susceptible as Nebraska 145-4. Cimarron appeared to be the most resistant variety. Anjou, Baker 195, Illinois 48-36, and Western Oil Hybrid 9 appeared to be in the same class and may be somewhat resistant. Baker 1, Illinois 48-84, Nebraska Hybrid 1, USDA 74, USDA 101, USDA 144, and Western Oil Hybrid 4 were intermediate in their reaction. The Conner and USDA 9/19 varieties were quite susceptible but not so much so as Nebraska 145-4.

Literature Cited

1. Classen, Carl E., and Albert Hoffman. 1950. The inheritance of the pistillate character in castors and its possible utilization in the production of commercial hybrid seed. Agron. Jour. 42: 79-82.
2. Elliott, Charlotte. 1951. Manual of bacterial plant pathogens. 1951. Chronica Botanica Co., Waltham, Mass.
3. Patel, M. K., Y. S. Kulkarni, and G. W. Dhande. 1951. Bacterial leaf-spot of castor. Current Science 20: 20.
4. Van Horn, D. L., and L. H. Zimmerman. 1953. Performance of castorbean inbred lines and their hybrid combinations with Nebraska 145-4. TMSC-5 U. S. Department of Agriculture.

DIVISION OF TOBACCO, MEDICINAL AND SPECIAL CROPS, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, UNITED STATES DEPARTMENT OF AGRICULTURE AND THE TEXAS AND OKLAHOMA AGRICULTURAL EXPERIMENT STATIONS.

STRAINS OF SUGARCANE MOSAIC IN PUERTO RICO

George W. Bruehl[1]

Mosaic continues to be a major disease of sugarcane in Puerto Rico as shown by the results of a recent survey of the Island presented in Table 1. For many years the only susceptible variety grown was BH 10(12), in which the disease was kept to a low percentage where roguing and seed selection were done carefully. When B. 34104 became a commercial variety, with extensive acreages in the south and east coast areas, mosaic spread rapidly. The resultant build-up of the disease has led to infection of B. 37161, M. 336, and H. 32-8560. B. 34104 has withstood mosaic in several of the islands of the British West Indies (9, p. 42), but has become highly infected in Jamaica.

Commercial varieties in Puerto Rico should have a high degree of resistance to mosaic, and because of the importance of the disease, inoculation of seedling populations with the virus has become a part of the breeding and varietal improvement program. In order to know what should be used as inoculum in the routine progeny tests, a survey of mosaic strains on the island was undertaken. Previous knowledge of the strains of the virus in Puerto Rico is limited to the studies of Matz (5), who intercepted mosaic in the quarantine houses at Arlington Farm, Virginia, and found that the common mosaic of BH 10(12) from Puerto Rico corresponded to strain B of Louisiana (7), and the observation of Jensen (2) of symptoms on cane in commercial fields suggesting the presence of two or more strains.

Table 1. Mosaic observed in commercial fields in Puerto Rico, August-September, 1952.

Variety	Number of fields observed	Highest percent of mosaic encountered	Average percent infection of all fields observed
B. 34104	19	95	38
BH 10(12)	7	64	17
B. 37161	14	8[a]	2
H. 32-8560	10	5	1
M. 336	17	3[b]	0.2

[a] Higher infections have subsequently been observed near Caguas and Mercedita.
[b] Higher infections have subsequently been observed at Yabucoa with Mr. J. Adsuar of the Plant Pathology Department and Mr. Gonzalez-Rios, Head, Department of Plant Breeding, Puerto Rico Agricultural Experiment Station, and at Mercedita.

In the present study accessions of mosaic were collected from different areas and varieties. Stem cuttings from infected plants were grown in large clay pots in the greenhouse until large enough for juice extraction. In this initial screening process two differential hosts (8) were used, C. P. 31-294 and C. P. 29-291. Inoculation was by the Matz (4) method. Five plants of each differential were inoculated.

An attempt was made to relate the strains observed with those of continental United States, as shown in Table 2. Several of the accessions developed symptom patterns resembling strains A, B, C, or D, while some did not correspond to any descriptions given. For the most part these unknowns produced a milder pattern than is typical of strain A. These need further study. It is obvious, however, from this exploratory survey that the mosaic problem of Puerto Rico is complex, and that several strains of the virus are present here.

Plants 4-5 weeks old of the five varieties of cane found infected in commercial fields were then inoculated by the Sein leaf-slip method (6) with leaves of C. P. 31-294 representing groups

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Table 2. Type of mosaic observed on C.P. 31-294 approximately 60 days after inoculation, February-May, 1953, at Rio Piedras, Puerto Rico.

Origin of Accession / Area	Tentative strain of identification: A	B	C-D[a]	Unidentified	Total
Yabucoa		19	3	3	25
Humacao	1	5	1	2	9
Aguirre	1	4	2	2	9
Cortada	5	4	3	2	14
Mercedita	4	9			13
Caguas	1	6	3	4	14
Vega Alta			1		1
Fajardo		1			1
Total	12	48	13	13	86
Variety					
B. 34104	5	19	5	7	36
BH 10(12)	2	12	2	1	17
B. 37161	2	6	6	2	16
B. 4098		6			6
B. 41211	2	3			5
Unknown		1		2	3
H 32-8560		1			1
M. 336	1				1
Aguirre Seedling				1	1
Total	12	48	13	13	86

[a] It is not possible to distinguish C from D on the differential hosts used in this trial.

A, B, C or D. The varietal reaction obtained in this test corresponded roughly to observed infection in the field (Tables 1 and 3), agreeing with results with this technique in Hawaii (3). An important descrepancy lay in that strain B predominated in the field, but strain A was more infective and had a shorter incubation period in these pin-prick tests. Group C-D had the lowest infectivity except on B. 37161. H. 32-8560 appeared very resistant as few plants were infected. M. 336 had been considered a mosaic resistant variety for years, but Adsuar (1) reported mosaic on it, and the infection has been increasing. M. 336 was infected by all strains used in this test, so that the explanation probably does not rest upon the appearance of a new strain, but rather that the variety previously had escaped infection.

Literature Cited

1. Adsuar, Jose. 1950. Preliminary report of a mosaic disease of the resistant sugar cane variety Mayaguez-336. Univ. of Puerto Rico Agricultural Expt. Sta. Tech. Paper 7. 9 p., illus.
2. Jensen, James H. 1936. The present sugarcane disease situation in Puerto Rico. Agr. Notes, Mayaguez, Puerto Rico. No. 69.
3. Martin, J. P., and C. W. Carpenter. 1935. Testing cane varieties for disease

Table 3. Susceptibility of cane varieties to strains of sugarcane mosaic as shown by pin-prick, leaf slip inoculation, May-June, 1953, at Rio Piedras, Puerto Rico.

Variety	Number of plants		Percent Infected	Incubation period in days	
	Inoculated	Infected		Range	Mean
Strain A:					
B. 34104	31	30	96	11-20	16
BH 10(12)	35	32	91	10-21	16
M. 336	33	20	61	9-34	16
B. 37161	32	25	78	16-34	22
H. 32-8560	39	5	13	13-29	16
Strain B:					
B. 34104	34	23	68	16-27	18
BH 10(12)	26	14	54	19-27	20
M. 336	36	10	28	12-31	18
B. 37161	32	4	13	28-36	28
H. 32-8560	36	1	3		14
Strains C-D:					
B. 34104	27	11	41	17-22	18
BH 10(12)	34	11[a]	32	19-30	20
M. 336	32	5	16	15-25	22
B. 37161	32	4	13	14-20	16
H. 32-8560	35	0	0		

[a]Infection was obtained with 6 out of 9 accessions of C-D inoculated to BH 10(12). Strains A, B, C-D appeared similar on this variety. E. V. Abbott of the Division of Sugar Plant Investigations, U. S. Department of Agriculture, in correspondence, stated that strains A, B, and D are indistinguishable and produce a mild pattern on BH 10(12), but that strain C produces a severely chlorotic and necrotic pattern. On this basis the C-D accessions transferred to BH 10(12) would all be D.

resistance in Hawaii. Proc. Int. Soc Sugar Cane Tech. 5th Congress, Brisbane, Australia. pp. 519-521.

4. Matz, J. 1933. Artificial transmission of sugarcane mosaic. Jour. Agr. Res. 46: 821-839. illus.
5. _____. 1938. Comparative study of sugarcane mosaic from different countries. Proc. Internat. Soc. Sugar Cane Tech., 6th Congress. pp. 572-580. illus.
6. Sein, F., Jr. 1932. Artificial transmission and other studies on sugar cane mosaic. Proc. Int. Soc. Sugar Cane Tech. 4th Congress, San Juan, Puerto Rico. Bull. 84. 6 pp.
7. Summers, E. M. 1936. An investigation of types or strains of the mosaic virus of sugarcane in Louisiana. Iowa State Coll. Jour. Sci., 11: 118-120.
8. _____, E. W. Brandes and R. D. Rands. 1948. Mosaic of sugarcane in the United States, with special reference to strains of the virus. U. S. Dept. Agri. Tech. Bull. 955. 124 pp., illus.
9. Turner, P. E. 1952. Report of research work on sugar-cane agriculture in the British West Indies. 1950-51, 58 pp. Publication of the British West Indies Sugar Association, Port-of-Spain, Trinidad, B. W. I.

JOINT CONTRIBUTION FROM THE DIVISION OF SUGAR PLANT INVESTIGATIONS, U.S. DEPARTMENT OF AGRICULTURE, AND THE PUERTO RICO AGRICULTURAL EXPERIMENT STATION

PYRETHRUM COLLAR ROT IN PERU[1]

Bowen S. Crandall and Javier Diéguez C.[2]

Pyrethrum, Chrysanthemum cinerariaefolium Trev., was introduced into Peru in 1928; but, for the next 14 years, little was done with the crop. Not until after World War II broke out was the crop given much impetus, and then several diseases to which it was susceptible came into prominence.

In the lowlands, at the Estación Experimental Agrícola in the Tingo María area (elevation, 2,200 feet), introductions of pyrethrum were uniformly unsuccessful. Plants died as a result of a root and collar rot caused by Rhizoctonia solani Kuehn.

In the highlands, where a program of experimentation with the crop was carried on at the Estación Experimental Agrícola de Junin at Concepción near Huancayo (elevation, 13,000 feet), another collar rot was observed. It was first seen there in 1937 and reported by Ing. Ocampo in the 1937 report of the Estación Experimental de La Molina. The same disease was observed by Crandall and Martin and also by Culbertson in 1943 and mentioned in reports to the Office of Foreign Agricultural Relations, U. S. Department of Agriculture.

All observers mentioned the connection between incidence of the disease and use of an improper irrigation practice. Basically, this practice consisted of setting the young plants in the bottom of a furrow to facilitate irrigation at the time of transplanting. As the plants grew, the furrow was gradually closed in and a new furrow opened between the rows for irrigating the mature plants. This practice resulted in covering a considerable part of the crown of the pyrethrum plant. A dark-brown firm decay developed in this covered part; and the invaded tissue involved the leaf bases, the collar, and the adjoining parts of the roots. A disease with similar symptoms developed in seedbeds that were irrigated by flooding.

In November 1946 the disease was briefly investigated, and material was brought to Tingo María for isolation of the pathogen. Almost all the cultures prepared from leaf-base, collar, and root material yielded a strain of Phytophthora cactorum (Leb. & Cohn) Schroet. This strain produced sporangia 37.4-51 x 23.8-27.2 microns and chlamydospores 30-37.5 microns. No oogonia were observed in the cultures. The fungus grew well at temperatures just below 40° F and was not injured by freezing.

A similar disease of pyrethrum, which appeared in India in 1939-46, has been reported by Ramakrishnan and Soumini (Indian Phytopath. 1: 27-33. 1948). They found the causal organism to be Phytophthora cambivora (Petri) Buis. Their description and text figures indicate that their identification is correct and that the Indian and Peruvian diseases, through similar in symptoms, are caused by different species of Phytophthora.

ESTACIÓN EXPERIMENTAL AGRÍCOLA EN TINGO MARÍA, PERU

[1]A contribution from the Estación Experimental Agrícola en Tingo María, a technical agricultural service organization from the Orient of Peru, operated jointly by the Ministry of Agriculture of Peru and the Foreign Agricultural Service of the U. S. Department of Agriculture. United States participation in this work was carried out as part of the Point IV program in Peru, administered by the Technical Cooperation Administration, U. S. Department of State, through the Institute of Inter-American Affairs.

[2]Mr. Crandall is Research Advisor in Pathology, Foreign Agricultural Service, U. S. Department of Agriculture, now assigned to the cooperative fiber project in Cuba and formerly Chief, Department of Plant Pathology, Estación Experimental Agrícola en Tingo María, Peru; Mr. Diéguez is Chief, Department of Plant Pathology and Entomology, Estación Experimental Agrícola en Tingo María.

A PRELIMINARY NOTE ON THE SUSCEPTIBILITY OF WOUNDED OAKS TO NATURAL INFECTION BY THE OAK WILT FUNGUS

F. C. Craighead[1], C. L. Morris, and J. C. Nelson

Observations made by workers in several States (2, 3) indicate that numerous insects in all stages of development are invariably associated with the sporulating mats of the oak wilt fungus (Endoconidiophora fagacearum) when they are produced on diseased trees. The importance of these insects in the probable diploidization of the fungus was noted by Leach et al. (4), and Curl and Zuckerman (2). Attention was called to their possible importance as vectors of the disease by Craighead and Morris (1).

To investigate the importance of man-made wounds in the spread of the disease, a replicated series of wounds of various types was initiated in September 1952. Additional series of wounds were established during the spring and summer of 1953. Wound plots were established on three widely separated areas where mycelial mats were being produced on nearby diseased trees. These mats were expected to produce the necessary inoculum for possible infection of these wounded trees. All wounded trees were located at least 300 yards from the nearest mat-producing tree except in one series where bolts of diseased oak bearing numerous fresh mats and fertile perithecia were distributed within 50 feet of the wounded trees 1, 2, and 3 in Area A (see Table 1).

To determine what effect the time factor would have on the susceptibility of wounds, the wounds were covered approximately one week after being made, at which time additional replications of the wound series were added. A total of 194 trees have been wounded periodically from March 23 to July 31, 1953.

On July 16 seven trees (four in one area and three in the second) were found to be wilting. An additional tree was added to this list on July 28. Laboratory cultures confirmed infection. No wounded trees on the third plot have exhibited wilting symptoms to date. Table 1 summarizes this information.

Table 1. Data concerning trees that became infected by the oak wilt fungus in wounding experiments in Pennsylvania.

Area No.	Tree No.	Date wounded	Date covered	Period wound open (days)	Type of wound	Date fertile perithecia noted nearby
A	1	May 11	May 20	9	Bark bruise[a]	May 10
	2	"	"	9	Drill holes (2)	"
	3	"	"	9	Bark bruise	"
	4	May 20	June 2	13	Drill holes (2)	"
	5	"	"	13	Drill holes (2)	"
B	1	May 12	May 21	9	Bark bruise	On May 13, 5 bolts of oak bearing mats with perithecia were distributed near trees wounded in this series.
	2	"	"	9	Bark bruise	
	3	"	"	9	Axe cuts	
C	No trees wilting to date in this plot					

[a] All wounds made on the lower bole.

When wounds on wilted trees were uncovered and the insects collected from the wounds, larvae of several species of Nitidulids and Diptera were abundantly represented. These same

[1] Consulting Entomologist Pennsylvania Dept. of Agriculture.

insects were found associated with fresh sporulating mats of the fungus. A complete list of the insects associated with these wounds and with the mycelial mats in Pennsylvania will be published at a later date.

It will be noted that those wounded trees in the two areas which became infected with oak wilt were all in the near vicinity of diseased trees on which mycelial mats bearing fertile perithecia were produced. In the third area only mats bearing conidiospores were observed as a source of inoculum. It seems significant that on both areas the date of known production of ascospores coincided closely with the period during which wounds were open on nearby trees that later wilted.

Literature Cited

1. Craighead, F. C., and C. L. Morris. 1952. Possible importance of insects in transmission of oak wilt. Pennsylvania Forests and Waters Nov.-Dec. 1952.
2. Curl, E. A., and B. M. Zuckerman. 1953. Sub-cortical mycelial mats and perithecia of the oak wilt fungus in nature. Phytopath. 43: 65-70.
3. Leach, J. G., R. P. True, and C. K. Dorsey. 1952. A mechanism for liberation of spores from beneath the bark and for diploidization of Chalara quercina. Phytopath. 42 (10): 537-539.
4. _____, C. K. Dorsey, R. P. True, and H. L. Barnett. 1952. Insects and the oak wilt fungus. Science, Agric. Exp. Sta., West Virginia Univ. Bull. 357.

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SEED-BORNE PLANT PATHOGENS

E. L. LeClerg[1]

Seed pathology has received serious attention in its relation to the quality of agricultural seeds. Much progress has been made in the development of methods for detecting seed-borne plant pathogens but most of them are still too time-consuming to be practical as a routine part of a seed certification program. Among the factors that hinder the general application of seed pathology to the determination of clean seed are the lack of satisfactory laboratory methods for rapid detection of seed-borne pathogens, scarcity of information as to the correlation of laboratory tests on contaminated seed with subsequent field response of crops, and want of personnel trained in the identification of seed-borne pathogens.

The following list includes plant pathogens borne on or in true seed, or with bulbs, corms, seed tubers, etc.[2].

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[1]Research Coordinator, Agricultural Research Administration, U. S. Department of Agriculture, Washington, D. C.

The writer wishes to express his thanks to the many plant pathologists who assisted in the preparation of the list of seed-borne plant pathogens. The list was compiled as part of an article for the 1953 U. S. D. A. Yearbook to indicate the extent of the problem.

[2][Editor's note]: Reference should be made to the various lists and technical articles on seed-borne parasites, especially the following:

International Seed Testing Association. List of seed-borne plant pathogens to be published as an appendix to the forthcoming International Rules for seed testing.

Groves, J. W., and A. J. Skolko. Notes on seed-borne fungi. I. Stemphylium. II. Alternaria. III. Curvularia. Can. Jour. Res., C, 22: 190-199, 217-234. 1944;23:94-104. 1945. (and others in a series).

Neergaard, Paul. Seed-borne fungous diseases of horticultural plants. Compt. Rend. Assoc. Intern. Essais Semences. Stockholm, 1940, pp. 47-71.

_____. Danish species of Alternaria and Stemphylium. Taxonomy, parasitism, economical significance. Communication from the Phytopathological Laboratory of J. E. Ohlsens Enke, Copenhagen. 559 pp. Einar Munksgaard, Copenhagen;Oxford University Press, London. 1945.

Orton, C. R. Seed-borne parasites. A bibliography. West Virginia Agr. Exp. Sta. Bull. 245. 1931.

SEED-BORNE PLANT PARASITES[3]

Host	Disease	Cause
ALFALFA Medicago sativa	Downy mildew	Peronospora trifoliorum (E)[4]
	Leaf spot	Leptosphaeria pratensis (I)
	Spring black stem	Ascochyta imperfecta (I, E)
	Summer black stem	Cercospora zebrina (I)
BARLEY Hordeum vulgare	Bacterial blight, black chaff	Xanthomonas translucens (E)
	Black loose smut	Ustilago nigra (E)
	Covered smut	Ustilago hordei (E)
	False stripe	False stripe virus (I)
	Fusarium blight	Gibberella zeae (I)
	Loose smut	Ustilago nuda (I)
	Net blotch	Pyrenophora teres (I)
	Septoria blotch	Septoria nodorum (I)
	Spot blotch and crown rot	Helminthosporium sativum (I, E)
	Stripe	Helminthosporium gramineum (I, E)
BEAN Phaseolus vulgaris	Anthracnose	Colletotrichum lindemuthianum (I, E)
	Bacterial blight	Corynebacterium flaccumfaciens (I)
	Bacterial blight	Xanthomonas phaseoli (I)
	Common bean mosaic	Marmor phaseoli, Phaseolus virus 1, (I)
	Fuscous blight	Xanthomonas phaseoli var. fuscans (I)
	Halo blight	Pseudomonas phaseolicola (I)
	Mosaic	Bean virus 4, Marmor laesiofaciens (I)
		New York 15 virus strain of Marmor phaseoli (I)
	Stem anthracnose	Colletotrichum truncatum (E)
BEET Beta vulgaris, including SUGAR BEET	Bacterial blight	Pseudomonas aptata (E)
	Downy mildew	Peronospora schachtii (I, E)
	Leaf spot	Cercospora beticola (E)
	Root disease	Phoma betae (E)
BLUEGRASS Poa pratensis	Leaf spot	Helminthosporium vagans (E)
BROMEGRASS Bromus species	Head smut	Ustilago bullata (E)
	Leaf spot	Selenophoma bromigena (I)
BRUSSELS SPROUTS Brassica oleracea gemmifera	Blackleg	Phoma lingam (I)
	Black rot	Xanthomonas campestris (I)
	Leaf spot	Alternaria brassicae and A. brassicicola (E)
BUR-CLOVER Medicago hispida	Leaf spot	Cercospora medicaginis

[3]Includes parasites carried on the surface of the seed coat, pericarp, or under floral bracts or in the seed coat, pericarp, cotyledons, or embryo; or in bulbs, tubers, etc.

[4]I, Internally borne; E, Externally borne.

Host	Disease	Cause
CABBAGE Brassica oleracea capitata	Blackleg Black rot Leaf spot	Phoma lingam (I) Xanthomonas campestris (I) Alternaria brassicae and A. brassicicola (E)
CALLA LILY RHIZOMES Zantedeschia species	Rhizome rot	Erwinia aroideae, E. carotovora, Phytophthora erythroseptica, P. richardiae (I)
	Tomato spotted wilt virus	Lethum australiense, Lycopersicon virus 3 (I)
CARROT Daucus carota	Bacterial blight Black rot Cercospora blight	Xanthomonas carotae (E) Stemphylium radicinum (E) Cercospora carotae (E)
CAULIFLOWER Brassica oleracea botrytis	Alternaria blight Blackleg Black rot Leaf spot	Alternaria dauci (I) Phoma lingam (I) Xanthomonas campestris (I) Alternaria brassicae and A. brassicicola (E)
CELERY Apium graveolens	Late blight	Septoria apii (I)
CLOVERS Trifolium species	Black stem Downy mildew Southern anthracnose	Cercospora zebrina (I) Peronospora trifoliorum (I) Colletotrichum trifolii (I)
CORN Zea mays, including sweet corn	Bacterial wilt Black kernel blight Ear and stalk rot Seedling blight	Bacterium stewartii (I, E) Helminthosporium carbonum (I) Diplodia zeae and D. macrospora (E) Diplodia zeae, Gibberella fujikuroi, G. zeae, Nigrospora oryzae, Penicillium oxalicum (I, E)
COTTON Gossypium species	Angular leaf spot and blight Anthracnose Seedling blight Seedling blight Wilt	Xanthomonas malvacearum (I, E) Glomerella gossypii (I) Ascochyta gossypii and Fusarium moniliforme (E) Glomerella gossypii (I) Fusarium oxysporum f. vasinfectum (E), Verticillum albo-atrum (I, E)
COWPEA Vigna cylindrica	Wilt	Fusarium oxysporum f. tracheiphilum (E)
CUCUMBER Cucumis sativus	Angular leaf spot Anthracnose Mosaic Scab	Pseudomonas lachrymans (I, E) Colletotrichum lagenarium (I) Marmor cucumeris, Cucumis virus 1 (I) Cladosporium cucumerinum (E)

Host	Disease	Cause
DAHLIA ROOTS Dahlia species	Cucumber mosaic virus	Marmor cucumeris, Cucumis virus 1 (I)
	Dahlia mosaic	Marmor dahliae, Dahlia virus 1 (I)
	Tomato spotted wilt virus	Lethum australiense, Lycopersicon virus 3 (I)
EGGPLANT Solanum melongena	Blight	Phomopsis vexans (E)
FESCUE Festuca species	Net blotch	Helminthosporium dictyoides (I)
FLAX Linum usitatissimum	Anthracnose	Colletotrichum lini (I, E)
	Pasmo	Mycosphaerella linorum (E)
	Stem break	Polyspora lini (I)
	Wilt	Fusarium oxysporum f. lini (E)
GARLIC BULBS Allium sativum	Yellow dwarf	Marmor cepae, Allium virus 1 (I)
GLADIOLUS CORMS Gladiolus species	Aster yellows virus	Chlorogenus callistephi, Callistephus virus 1 (I)
	Bean yellow mosaic virus	Phaseolus virus 2 (I)
	Blight	Botrytis gladiolorum, Curvularia lunata (I)
	Cucumber mosaic virus	Marmor cucumeris, Cucumis virus 1 (I)
	Dry rot	Stromatinia gladioli (I)
	Hard rot and leaf spot	Septoria gladioli (I)
	Rot	Fusarium oxysporum f. gladioli (I)
	Scab	Pseudomonas marginata (I)
	Tobacco ring spot virus	Annulus tabaci, Nicotiana virus 12 (I)
	Western aster yellows virus	Chlorogenus callistephi var. californicus, Callistephus virus 1A (I)
HAIRY VETCH Vicia villosa	Blight	Ascochyta pisi (I)
HYACINTH BULBS Hyacinthus orientalis	Soft rot	Erwinia carotovora (I)
	Yellows	Xanthomonas hyacinthi (I)
IRIS BULBS Iris species	Crown rot	Sclerotium rolfsii and S. delphinii (I, E)
	Mosaic	Marmor iridis, Iris virus 1 (I)
	Soft rot	Erwinia carotovora (I)
KALE AND COLLARDS Brassica oleracea viridis	Blackleg	Phoma lingam (I)
	Black rot	Xanthomonas campestris (I)
	Leaf spot	Alternaria brassicae and A. brassicicola (E)

Host	Disease	Cause
KOHLRABI	Blackleg	Phoma lingam (I)
Brassica oleracea gongylodes	Black rot	Xanthomonas campestris (I)
	Leaf spot	Alternaria brassicae and A. brassicicola (E)
LESPEDEZA		
Lespedeza species	Bacterial wilt	Xanthomonas lespedezae (I)
LETTUCE	Anthracnose	Marssonina panattoniana (E)
Lactuca sativa	Mosaic	Marmor lactucae, Lactuca virus 1 (I)
LIMA BEAN	Bacterial blight	Xanthomonas phaseoli (I, E)
Phaseolus lunatus	Downy mildew	Phytophthora phaseoli (I)
	Halo blight	Pseudomonas phaseolicola (I, E)
LILY BULBS	Black scale	Colletotrichum lilii (I)
Lilium longiflorum	Bulb rot	Fusarium oxysporum f. lilii (I)
	Cucumber mosaic virus	Marmor cucumeris, Cucumis virus 1 (I)
	Rosette	Virus (Lilium virus 1) (I)
	Tulip-breaking virus	Marmor tulipae, Tulipa virus 1 (I)
LUPINE	Anthracnose	Glomerella cingulata (I, E)
Lupinus species	Brown spot	Ceratophorum setosum (I)
	Mosaic	Pea mosaic viruses (I)
MILLET	Downy mildew	Sclerospora graminicola (I)
Chaetochloa italica and	Head smut	Sphacelotheca destruens (E)
Panicum miliaceum	Kernel smut	Ustilago crameri (E)
MUSKMELON	Angular leaf spot	Pseudomonas lachrymans (E)
Cucumis melo	Anthracnose	Colletotrichum lagenarium (I)
	Mosaic	Squash mosaic virus (I)
NARCISSUS BULBS	Bulb rot	Fusarium oxysporum f. narcissi (I)
Narcissus species	Mosaic	Narcissus mosaic virus (I)
	Streak	Narcissus streak virus (I)
OATS	Bacterial halo blight	Pseudomonas coronafaciens (E)
Avena sativa	Bacterial stripe blight	Pseudomonas striafaciens (E)
	Black loose smut	Ustilago avenae (E)
	Blotch	Pyrenophora avenae (I)
	Covered smut	Ustilago kolleri (E)
	Fusarium blight	Gibberella zeae (I)
	Leaf spot	Helminthosporium avenae (E)
	Loose smut	Ustilago avenae (E)
	Victoria blight	Helminthosporium victoriae (E)
ONION BULBS	Aster yellows	Chlorogenus callistephi, Callistephus virus 1 (I)
Allium cepa	Downy mildew	Peronospora destructor (I)
	Neck rot	Botrytis allii (I)
	Pink root	Pyrenochaeta terrestris (I)

Host	Disease	Cause
	Smudge	Colletotrichum circinans (E)
	Smut	Urocystis cepulae (E)
	Western aster yellows	Chlorogenus callistephi var. californicus, Callistephus virus 1A
	Yellow dwarf	Marmor cepae, Allium virus 1 (I)
PEA, GARDEN Pisum sativum	Anthracnose	Colletotrichum pisi (E)
	Bacterial blight	Pseudomonas pisi (I)
	Blight	Mycosphaerella pinodes (I)
	Leaf and pod spot	Ascochyta pisi (I)
	Septoria blight	Septoria pisi (E)
PEA, FIELD Pisum sativum var. arvense	Black stem	Ascochyta pinodella and Mycosphaerella pinodes (I)
	Downy mildew	Peronospora viciae (E)
PEPPER Capsicum annum	Anthracnose	Gloeosporium piperatum (I)
	Bacterial spot	Xanthomonas vesicatoria (E)
	Blight	Phytophthora capsici (I)
PETUNIA Petunia hybrida	Tobacco ring spot virus	Annulus tabaci, Nicotiana virus 12 (I)
POTATO TUBERS Solanum tuberosum	Black dot	Colletotrichum atramentarium (E)
	Blackleg	Erwinia phytophthora (I)
	Black wart	Synchytrium endobioticum (E)
	Brown rot	Xanthomonas solanacearum (I)
	Calico	Marmor medicaginis var. solani, Solanum virus 10 (I)
	Late blight	Phytophthora infestans (I)
	Latent mosaic	Annulus dubius, Solanum virus 1 (I)
	Leaf roll	Corium solani, Solanum virus 14 (I)
	Mild mosaic	Marmor solani, Solanum virus 3 (I)
	Net necrosis	Corium solani, Solanum virus 14 (I)
	Powdery scab	Spongospora subterranea (E)
	Rhizoctoniose	Rhizoctonia solani (Pellicularia filamentosa) (E)
	Ring rot	Corynebacterium sepedonicum (I)
	Rugose mosaic	Annulus dubuis plus Marmor upsilon, Solanum virus 2 (I)
	Scab	Actinomyces scabies (E)
	Silver scarf	Spondylocladium atrovirens (E)
	Spindle tuber	Acrogenus solani var. vulgaris, Solanum virus 12 (I)
	Vein banding mosaic	Marmor upsilon, Solanum virus 2 (I)
	Wilt	Fusarium oxysporum (I), Verticillium albo-atrum (I, E)
	Witches'-broom	Chlorogenus solani, Solanum virus 15 (I)

Host	Disease	Cause
	Yellow dwarf	Aureogenus vastans, Solanum virus 16 (I)
RADISH Raphanus sativus	Black leaf spot	Alternaria raphani (E)
	Blackleg	Phoma lingam (I)
	Black rot	Xanthomonas campestris (I)
	Bacterial spot	Xanthomonas vesicatoria var. raphani (E)
RED CLOVER Trifolium pratense	Anthracnose	Kabatiella caulivora (E)
	Black patch	Undetermined fungus (E)
	Downy mildew	Peronospora trifoliorum (E)
RICE Oryza sativa	Blast	Piricularia oryzae (I)
	Brown spot	Helminthosporium oryzae (I)
	Cercospora spot	Cercospora oryzae (I)
	Kernel smut	Neovossia horrida (E)
RYE Secale cereale	Bacterial blight, black chaff	Xanthomonas translucens f. sp. undulosa (E)
	Fusarium blight	Gibberella zeae (I)
	Stalk smut	Urocystis occulta (E)
RYEGRASS Lolium species	Blind seed	Phialea temulenta (I)
SHALLOT BULBS Allium ascalonicum	Aster yellows	Chlorogenus callistephi, Callistephus virus 1 (I)
	Yellow dwarf	Marmor cepae, Allium virus 1 (I)
SORGHUM Sorghum vulgare	Bacterial streak	Xanthomonas holcicola (E)
	Bacterial stripe	Pseudomonas andropogoni (E)
	Covered kernel smut	Sphacelotheca sorghi (E)
	Fusarium blight	Gibberella zeae (I)
	Holcus spot	Pseudomonas syringae (E)
	Leaf blight	Helminthosporium turcicum (I)
	Loose kernel smut	Sphacelotheca cruenta (E)
SPINACH Spinacia oleracea	Downy mildew	Peronospora effusa (E)
SQUASH Cucurbita maxima	Mosaic	Squash mosaic
	Root rot	Fusarium solani f. cucurbitae (I)
SUDANGRASS Sorghum vulgare var. sudanense	Anthracnose	Colletotrichum graminicola (I)
	Bacterial eye spot	Pseudomonas syringae (E)
	Bacterial streak	Xanthomonas holcicola (E)
	Bacterial stripe	Pseudomonas andropogoni (E)
	Covered kernel smut	Sphacelotheca sorghi (E)
	Leaf blight	Helminthosporium turcicum (I)
	Loose kernel smut	Sphacelotheca cruenta (E)
SUGARCANE CUTTINGS Saccharum officinarum	Chlorotic streak	Fractilinea quarta (I)
	Downy mildew	Sclerospora sacchari (I)
	Fiji disease	Galla fijiensis, Saccharum virus 2

Host	Disease	Cause
	Gummosis	Xanthomonas vasculorum (I)
	Leaf scald	Xanthomonas albilineans (I)
	Mosaic	Marmor sacchari, Saccharum virus 1 (I)
	Red rot	Physalospora tucumanensis (I)
	Smut	Ustilago scitaminea (I)
	Streak	Fractilinea maidis var. sacchari (I)
SWEETCLOVER Melilotus species	Black stem	Ascochyta caulicola (I)
	Leaf spot	Leptosphaeria pratensis (I)
	Spring black stem	Mycosphaerella lethalis (I)
	Summer black stem	Mycosphaerella davisii (I)
SWEET POTATO ROOTS Ipomoea batatas	Black rot	Endoconidiophora fimbriata (E)
	Internal cork	Undetermined virus
	Scurf	Monilochaetes infuscans (E)
	Stem rot	Fusarium oxysporum f. batatas (I)
TOBACCO Nicotiana tabacum	Blackfire or angular leaf spot	Pseudomonas angulata (E)
	Frogeye spot	Cercospora nicotianae (E)
	Tobacco ringspot virus	Annulus tabaci, Nicotiana virus 12 (I)
	Wildfire	Pseudomonas tabaci (E)
TOMATO Lycopersicon esculentum	Bacterial spot	Xanthomonas vesicatoria (E)
	Canker	Corynebacterium michiganense (E)
	Mosaic, tobacco mosaic virus	Marmor tabaci, Nicotiana virus 1 (E)
TULIP BULBS Tulipa species	Blight or fire	Botrytis tulipae (I, E)
	Gray bulb rot	Rhizoctonia tuliparum (I, E)
	Tobacco necrosis virus	Marmor lethale, Nicotiana virus 11 (I)
	Tulip-breaking virus	Marmor tulipae, Tulipa virus 1 (I)
TURNIP Brassica rapa	Bacterial spot	Xanthomonas vesicatoria var. raphani (E)
	Blackleg	Phoma lingam (I)
	Black rot	Xanthomonas campestris (I)
	Leaf spot	Alternaria brassicae and A. brassicicola (E)
WATERMELON Citrullus vulgaris	Anthracnose	Colletotrichum lagenarium (I)
	Gummy stem blight	Mycosphaerella citrullina (E)

AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE

BRIEF NOTES

FAILURE OF FALL APPLICATIONS TO CONTROL PEACH BACTERIAL SPOT

By H. W. Anderson

Small scale experiments in 1951-52 indicated that fall applications of copper sulfate if applied at the proper time would eliminate the source of primary infection for peach bacterial spot (Xanthomonas pruni) in the spring. Results obtained by Mason in Arkansas during two seasons were definitely encouraging. Both in Arkansas and in Illinois at Urbana the most favorable time of application seemed to be about the middle of October. If applied earlier some twig injury resulted while if applied as late as November 15 only about a 50 percent reduction in spring canker development resulted. It was realized that an entire orchard or large blocks of trees would have to be sprayed in order to reduce the chance of secondary infection from neighboring untreated trees.

In 1952, two large commercial orchards were selected for this test in Illinois. In addition, a solid block of peach trees consisting of some 30 varieties was selected at the Experiment Station at Urbana. All three of these orchards had a fairly severe leaf infection in 1952. The time of application was the middle of October, based on the results of the previous year and those of Mason in Arkansas in two seasons. Since Mason had found that a lime spray applied within an hour after the copper sulfate reduced the chance of injury but gave equal control most of the commercial orchards were sprayed with this safener. At Urbana a number of trees were sprayed with copper sulfate alone.

The results secured in the 1953 season were extremely disappointing. In none of the experiments was there any evidence of reduction of spring canker development nor of primary infection on the foliage over that found in the unsprayed trees.

It was found that fruit set was not reduced by the fall applications and very little twig injury occurred where copper sulfate was used without the safener. Leaf curl (Taphrina deformans) was not completely controlled but was reduced to the extent that it was of no importance.

It is hard to explain the failure of this promising method of bacterial spot control. Unfortunately no time interval applications were made this year and it is possible that the time selected was not favorable. It was noticed last summer that growth of terminals ceased much earlier than usual and the early maturing of the twigs may have protected the over-wintering bacteria.

Bacterial spot in Illinois is very light on both foliage and fruit in the commercial orchards mainly because of dry weather in the peach growing areas. This makes the evaluation of the treatments more difficult from a statistical standpoint.

I am inserting this note without data at this time since I know that a number of pathologists are interested in the control of bacterial spot and the encouraging results in the past may lead them to recommend the fall applications of copper sulfate. It is evident that more extensive investigations are required to determine the conditions under which control can be expected. I am still confident that the basic concept of control by fall application is sound.

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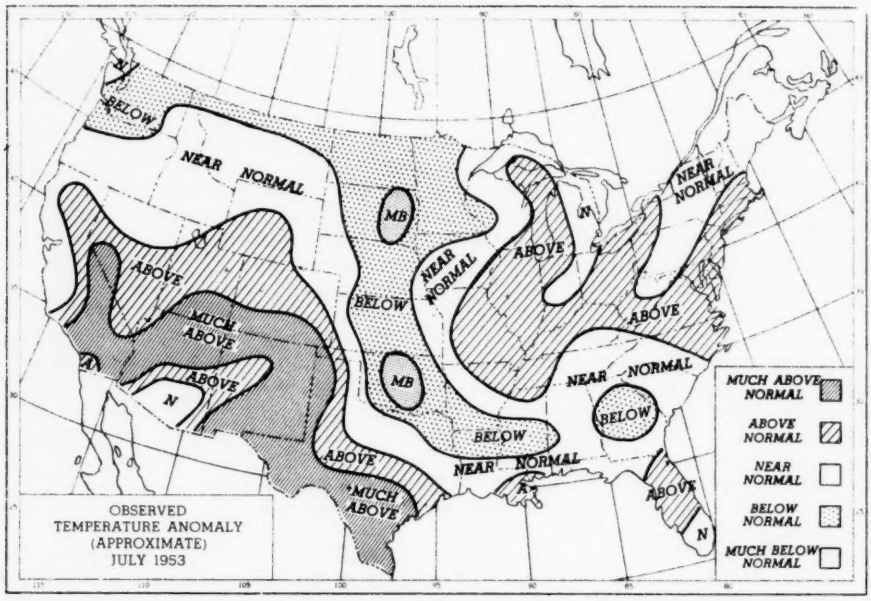
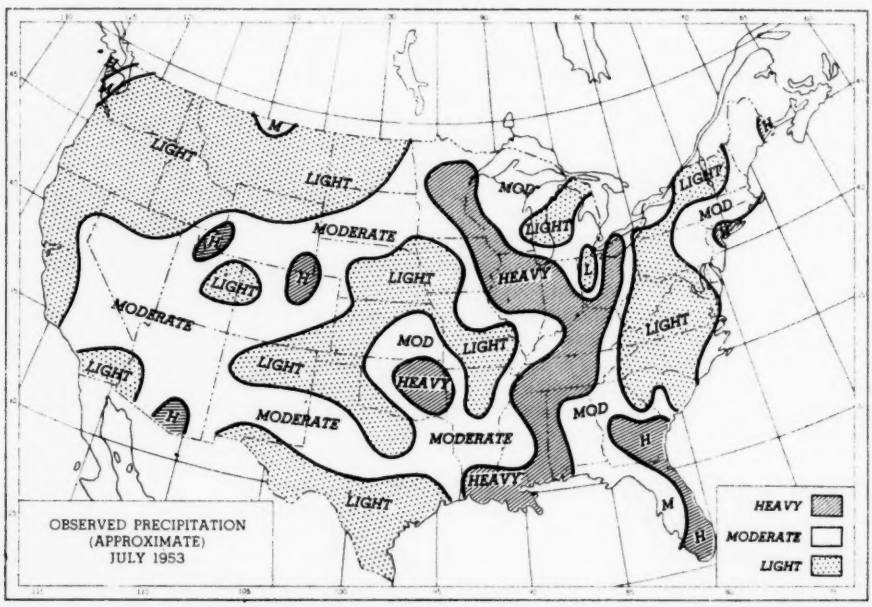
CORRECTION

On page 423 in the August 15 issue, in the article by V. R. Wallin and A. J. Skolko on inactivation of antifungal antibiotics by chlorine, the third line of the title for Figure 2 should obviously begin with "E -- H" instead of E-F as given.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

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